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Medical Management of Osteoarthritis

Case Study and Commentary:
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DR. LIANG:

Osteoarthritis (OA) is a common, chronic, progressive disease generally involving joints and surrounding tissue. In the United States, OA affects at least 6% of adults older than 30 years and 50% of adults older than 60 years.[1,2] Indeed, OA is the leading cause of disability reported in the United States, with significant costs associated both with direct medical and surgical interventions and with absence from work by those affected with the disorder.[3,4]

For primary care physicians, OA is a necessary diagnosis to consider in all patients reporting joint pain. OA is the reason for at least 2% of all visits to primary care physicians and is the 10th most frequent diagnosis encountered in that practice setting.[5] In light of the significant presence of OA in an elderly population, primary care physicians in the United States will most likely encounter more patients with this disorder as the US population ages.

The pathophysiology of OA is currently unclear. Generally, the smooth surface of hyaline cartilage becomes irregular, owing to changes on a cellular level and to mechanical forces.[1] However, the role of the inflammatory reaction at the joint site is not well understood but is instead the subject of significant debate. As the disease progresses, bone near the site changes, remodeling and creating additional irregularities that lead to narrowing of the affected joint space and, in some cases, chronic synovitis.[1] The joints most often affected include the knees, hips, cervical and lumbosacral spines, and distal interphalangeal (associated with Heberden's nodes), proximal interphalangeal (associated with Bouchard's nodes), and first carpometacarpal joints of the hand. Epidemiologic studies have pointed to genetic influences, at least for OA of the hip.[6,7]

In contrast to standard disease-assessment approaches that concentrate on a close attention to history, the diagnosis of OA is generally made on the basis of clinical findings and radiologic examination. Radio-graphic studies that suggest a diagnosis of OA include joint space narrowing, presence of osteophytes, irregular joint surfaces, sclerosis of subchondral bone, and bony cysts. However, these findings must be correlated with clinical symptoms.[8] Of course, in patients reporting joint pain, the physician should consider all possibilities in the differential diagnoses (eg, rheumatoid arthritis, gout, pseudogout, septic arthritis, tendonitis, bursitis).

Classification criteria for OA have been adopted by the American College of Rheumatology based on anatomic location (eg, knee,[9] hip,[10] hand[11]). Other classification schemes have also been proposed.[12]

Several pharmacologic treatment regimens are associated with symptom relief, as noted in the case studies presented in the following article. Specifically, nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, cyclooxygenase-2 (COX-2) inhibitors, glucosamines, and corticosteroids have all been reported to have some beneficial effect.[13] In addition, other, less traditional forms of treatment (eg, the use of leeches) have been proposed.[14] It is also worth noting that exercise can provide symptomatic relief in patients with mild to moderate pain. For example, patients report pain diminishment, reduced disability, and better walking performance and have improved results on global assessment of symptoms after undergoing physical therapy.[15,16]

Finally, patient education appears to be effective in assisting patients with OA to manage their disease, both in terms of pain control and the necessity for medical visits. More specifically, self-management programs such as

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the Arthritis Self-Management Program, which consists of 6 2-hour sessions, have been reported to decrease patient pain and the number of visits to physicians.[17] However, education may need to be given in a group setting; education regarding OA provided to patients during regularly scheduled visits was found to have no benefit and, indeed, may have resulted in worse physical function.[18]

In summary, OA is a common disorder in the United States. Increasingly, primary care providers are called on to make the diagnosis. Knowledge of the pharmacologic and physical therapies available is necessary to address the symptoms and to manage the potential joint degeneration associated with the disease. Educational efforts may need to focus on group interventions such as the Arthritis Self-Management Program; primary care providers should be well versed in the limits of office-based educational efforts in relieving pain and improving function.

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OA is the most common form of arthritis, affecting more than 1 in 10 Americans between the ages of 15 and 74 years.[19] OA can cause significant morbidity, translating into major expenditures in terms of direct and indirect costs of care.[20]

The belief that OA is an inevitable consequence of aging has been a barrier to developing and implementing effective therapies. Although this condition was once referred to as degenerative joint disease or "wear and tear" arthritis, OA is now known not to be an inevitable consequence of aging. Instead, OA is caused by a derangement of the entire joint that affects the subchondral bone, articular cartilage, menisci, joint capsule, and periarticular structures such as ligaments, tendons, and stabilizing muscles.

The pathogenesis of OA is the result of dynamic interplay of biochemical processes and biomechanical forces at the joint organ, ultimately resulting in focal degeneration of joint cartilage and new bone formation under the cartilage lesion (subchondral bone) and at joint margins (osteophytes).[21,22] Although OA was long considered the prototypical noninflammatory arthritis, it is now clear that inflammatory mediators (including cytokines, nitric oxide, and metalloproteases) play a role in OA. Research into the role of inflammation is ongoing, as investigators focus on developing the first disease/structure–modifying agents for OA.

CASE STUDY 1
Initial Presentation

A 57-year-old woman goes to her primary care physician because of right thumb and bilateral knee pain.

History

The pain in her thumb bothers her most during the workweek when she types on her computer. Her knee pain is notable with activities requiring knee bending (such as getting in and out of her car) and is more intense at the end of the day. The patient is an office manager who lives with her husband in a 2-story house; a few months ago, the couple moved their bedroom to the ground floor so that she would not have to use the stairs as often. This alleviated her knee pain significantly, but the improvement was only temporary.

Today, she describes her pain as constant and nagging in the late afternoon and evening. She "doesn't like to take medication" but has been taking ibuprofen every day for the past several weeks. Further questioning reveals that the patient has no significant past medical or surgical history other than well-controlled hypertension but has become very sedentary over the past few years and has gained about 30 lb. She has no drug allergies and is a nonsmoker.

- **What risk factors predispose this patient to OA?**

Age is a risk factor, but the single most important modifiable risk factor for OA is obesity.[23,24] Obesity in women has been linked to OA of the knees and hips,[25] and a link between weight and OA of the hand has also been proposed.[26] Mechanisms may include increase in body mass, altered biodynamics of gait,[27] genetic predisposition, or altered metabolism (eg, estrogens).

Metabolic correlates of OA have been investigated as well. Data from 1003 women between age 45 and 64 from the Chingford population study have suggested that hypertension, hypercholesterolemia, and diabetes may be associated with unilateral and bilateral OA of the knee, independent of age and body mass index (BMI).[28] The Baltimore Longitudinal Study of Aging cohort did not show these relationships when adjustments for age and obesity were made.[29] Deconditioning, with weakness of the quadriceps muscles relative to body weight, is another possible risk factor for OA of the knee.[30]

Physical Examination

Physical examination of the patient reveals a blood pressure of 140/90 mm Hg, a pulse of 80 bpm, a respiratory rate of 14 breaths/min, and a normal temperature. Her BMI is 31 (5′6″ tall, 187 lb). Head, neck, chest, cardiovascular, and abdominal examinations are unremarkable. Examination of her joints reveals bony, nontender enlargement of proximal and distal interphalangeal joints in both hands to varying degrees, without evidence of inflammation. There is apparent

"squaring" of the base of the first carpometacarpal (CMC) joint on the right hand, with tenderness to palpation. The patient has restricted range of motion at both hips but no pain or tenderness with movement. Both knees appear to have bony enlargement, with crepitus on movement. Her right knee is mildly painful with passive movement through its range of motion. Neither knee has an obvious effusion, but on closer examination the right knee is slightly warm and there is a positive bulge sign.

• **What are the physical findings of the joint with OA?**

In OA, common sites of involvement include the distal interphalangeal (DIP) and first CMC joints of the hand and the first metatarsophalangeal joint in the foot; also commonly involved are the hips, knees, and lumbar and cervical spines. Primary OA rarely involves the metacarpophalangeal joints, wrists, elbows, shoulders, or ankles. If synovitis (evidenced by swelling and warmth) is present, the joint may be tender.

Even without active inflammation, large joints (eg, hips, knees) may be painful with weight-bearing but nontender on examination. Joint enlargement occurs because of bony osteophytes, joint effusion, and synovial hyperplasia. As the disease progresses, there may be crepitus, gross deformity, and subluxation of the joint. Limited range of motion also ensues because of the variable contributions of muscle contractures, joint surface irregularities, or mechanical blockage by osteophytes or loose bodies ("joint mice").

Although she has no complaints about her hands, the patient has both Heberden's (DIP) and Bouchard's (proximal interphalangeal [PIP]) nodes, which are hypertrophic bone formations or spurs of the involved finger joints. They usually develop slowly and fairly asymptomatically over time, although clinical inflammation can occur in some patients. Gelatinous cysts can be the initial presentation. When the first CMC joint is involved, its range of motion becomes limited and a tender prominence may develop at the base of the first metacarpal bone, giving the hand a "squared-off" appearance. An erosive inflammatory variant of OA has been described that affects the interphalangeal joints of the hands.[31] Painful inflammatory episodes can lead to joint deformity and ankylosis.

Patients with OA of the knee often report problems with kneeling, climbing stairs, and getting on and off chairs. The knee joint should be examined for valgus or varus alignment, crepitus, and effusion. Individual osteophytes in affected joints may be felt as irregular bony masses. Quadriceps atrophy is frequently present. In advanced disease, joint instability may be present as a result of cartilage loss, with secondary effective lengthening of the collateral ligaments. In patellofemoral disease, pain is elicited by holding the patella firmly against the femur and instructing the patient to perform isometric contraction of the quadriceps.

In the evaluation of an effusion, large fluid collections with ballotable patellae are easily evident. If no effusion is obvious, the medial surface of the joint may be milked cephalad and laterally over the superior edge of the patella. The lateral side of the knee is then compressed; if a small amount of fluid is present, it will appear as a "bulge" on the medial side of the joint. This is the "bulge sign."

Diagnosis

Radiographs of the patient's knees show bilateral changes consistent with OA (medial compartment narrowing with small osteophytes and subchondral bony sclerosis), worse on the left than on the right. A diagnosis of OA is made. Since the knee pain is bothersome but not disabling, the physician recommends that the patient try to lose about 10 lb over the next 2 months. He also demonstrates leg extension exercises that she can perform to strengthen her quadriceps. In addition, he recommends continuing the NSAID for symptom relief and trying capsaicin cream 0.025%, applied 3 times daily to her knees. He tells her that the cream will take about 2 weeks to work and may initially cause burning and irritation.

• **Should this patient have been encouraged to use acetaminophen instead of an NSAID?**

Acetaminophen 1000 mg up to 4 times daily should be the first-line systemic analgesic treatment for most patients. This determination is based in part on the agent's perceived equivalent efficacy to NSAIDs but mostly on its favorable side-effect profile. (Heavy alcohol use and liver disease are the most notable contraindications to acetaminophen use.) In what is probably the best-known study in this area, Bradley et al[32] demonstrated the equivalence of acetaminophen and ibuprofen in most measures except for rest pain, where ibuprofen at both 1200 mg daily and 2400 mg daily was statistically superior to acetaminophen. Some recent studies have suggested that there may be a significant difference favoring NSAIDs in populations with moderate to severe pain from OA.[33] The 2000 update of the American College of Rheumatology guidelines for management of OA of the hip and knee allows for using NSAID therapy (or specific COX-2 inhibitors) first in patients with clinical presentations suggestive of inflammation.[34]

Table 1. Commonly Used NSAIDs

Acetic acids

Diclofenac (Voltaren, Cataflam)

Etodolac (Lodine)

Nabumetone (Relafen)

COX-2 inhibitors

Celecoxib (Celebrex)

Rofecoxib (Vioxx)

Meclofenamate (Meclomen)

Oxicams

Meloxicam (Mobic)

Piroxicam (Feldene)

Propionic acids

Ibuprofen (Advil, Motrin)

Naproxen (Naprosyn, Naprelan)

Oxaprozin (Daypro)

Salicylates

Aspirin

Diflusinal (Dolobid)

Salsalate (Salflex, Disalcid)

COX-2 = cyclooxygenase-2; NSAIDs = nonsteroidal anti-inflammatory drugs.

If acetaminophen and nonpharmacologic measures such as weight loss, temperature modalities, joint protection, and physical therapy do not provide enough symptom relief, a course of NSAIDs can be tried (**Table 1**). Indeed, NSAIDs are the most commonly prescribed agents for the treatment of both pain and inflammation in OA.[35] Given that NSAID-associated gastrointestinal (GI) toxicity is usually dose-related, the minimum effective dose for pain relief should be used. It is important to note that the NSAID dose required to achieve analgesia is typically lower than the dose needed for anti-inflammatory effect. If a patient requires frequent NSAID use or is at increased risk for NSAID toxicity because of age (older than 65 years), comorbid medical conditions (eg, cardiac disease, diabetes), concurrent use of oral glucocorticoids or anticoagulants, or prior history of peptic ulcer disease or gastrointestinal bleeding, interventions that lessen NSAID toxicity should be considered. The addition of a proton-pump inhibitor[36] or misoprostol[37] has been shown to reduce GI toxicity from NSAIDs.

The use of COX-2 inhibitors instead of traditional NSAIDs is another option that reduces adverse GI events. They have been shown to reduce the incidence of symptomatic and complicated upper GI adverse events when compared with NSAIDs.[38] COX-2 inhibitors (eg, celecoxib, rofecoxib) specifically inhibit the inducible COX-2 isoform that is generated in inflammatory states without significantly inhibiting the COX-1 isoform at therapeutic concentrations. COX-1 is constitutively expressed in many tissues, including the GI tract, where it is the key enzyme in the synthesis of GI-protective prostaglandins. For OA, the recommended dose of celecoxib is 200 mg daily or 100 mg twice daily; the recommended dose of rofecoxib is 12.5 to 25 mg daily. In the treatment of OA, COX-2 inhibitors have equivalent efficacy to nonselective NSAIDs.[39–41]

Capsaicin cream (available in 0.025% and 0.075% strengths), a local substance P depletor derived from the pepper plant, can be a useful topical agent.[42] Trials have been conducted mostly in OA of the hands.[43] Intra-articular corticosteroids or hyaluronate injections can be tried if a patient's knee does not respond favorably to systemic therapy. In the patient with advanced disease and intractable pain who neither is a surgical candidate nor wants to undergo surgery, narcotics can be used to relieve pain and restore quality of life.

- **Is it a concern if a patient's radiographic and clinical findings do not correlate?**

It is common for there to be a disparity between the clinical and radiographic findings in OA of the knee. In the case patient, the right knee, which was more symptomatic, had less severe radiographic findings than the left knee, which was only minimally symptomatic. The patient's symptoms rather than radiographic appearance should guide medical management.

Three Months Later

Three months after her initial visit, the patient returns to her physician's office. She has lost 10 lb and has improved but still has significant dull, aching knee pain related to activity. She has stopped using NSAIDs because she "heard they cause ulcers." Instead, she is taking a combination glucosamine sulfate/chondroitin sulfate product that a magazine recommended for arthritis pain. The patient asks her doctor whether he thinks this "natural" remedy will work.

- **Are either glucosamine sulfate or chondroitin sulfate effective in OA management?**

Most studies have evaluated these drugs alone; very little data exist on the combination. More studies are available on the use of glucosamine sulfate; they have been performed in Europe and suggest that the drug

can be as effective as 1200 mg daily of ibuprofen.[44] However, the onset of action appears to be much slower than that of NSAIDs. Two 3-year European double-blinded, placebo-controlled studies of OA of the knee have suggested that glucosamine sulfate may provide a small (0.1- to 0.3-mm) benefit compared with placebo in preserving knee joint space.[45,46] Results of a National Institutes of Health–sponsored OA trial with glucosamine sulfate, chondroitin sulfate, glucosamine/chondroitin combination, and placebo arms are yet to be reported.

At this time, the preparations that have been studied are thought to be safe and have variable efficacy in providing symptomatic relief from the pain of OA. Whether or not there is a "disease-modifying effect" remains controversial. Drawbacks to these products are the cost and the fact that different brands of glucosamine/chondroitin preparations may vary widely in their composition.

Follow-up

The patient is reassured that the "natural" remedy she is using is probably safe although of uncertain efficacy. At a follow-up visit 6 months later, the patient is still on a regimen of capsaicin cream, acetaminophen, and glucosamine/chondroitin and reports only minimal symptoms related to her knees. She has managed not to regain the 10 lb she lost 9 months ago and feels that the weight loss made the biggest difference in her knee pain.

CASE STUDY 2
Initial Presentation

A 71-year-old woman with severe OA of the knee presents to her internist for a follow-up visit. She is taking celecoxib 200 mg daily and has been receiving bilateral corticosteroid injections 3 to 4 times a year for the past few years. She also receives physical therapy. Despite these treatments, the patient continues to have knee pain that keeps her awake at night. Her internist refers her to a rheumatologist for further management.

- **What causes pain in the osteoarthritic joint?**

The pain of OA can arise from almost any component of the joint organ (**Table 2**). Damaged articular cartilage interferes with normal function of the joint and can lead to joint instability. In addition, an affected joint cartilage can become fragmented, and the fragments can stimulate an inflammatory response from the synovium. While this inflammation is generally less than that which accompanies the "inflammatory arthritides" such as rheumatoid arthritis, it may be significant enough to cause considerable pain.

Table 2. Etiologies of Pain in Osteoarthritis

Structure	Mechanism
Bursae	Inflammation
Cartilage	Inflammation, joint instability
Central nervous system	Psychological stress, sleep deprivation
Joint capsule	Stretch from joint distention
Ligaments	Stretch at insertions
Menisci	Tearing or degeneration
Muscle	Spasm, contractures
Osteophytes	Periosteal elevation, neural impingement
Subchondral bone	Bone ischemia, infarction
Synovium	Increased synovial fluid production

Torn menisci can also alter joint mechanics and further propagate cartilage degeneration by interfering with the smooth gliding of articular surfaces. Distention of the joint capsule by increased synovial fluid production is painful. Furthermore, a distended joint may have compromised venous blood flow because of increased intra-articular pressure. For this reason, arthrocentesis alone (even without glucocorticoid instillation) can be effective in relieving joint pain.

Supporting structures of the joint organ, including muscles (which can spasm in response to altered joint biomechanics) and surrounding bursae (which can become distended with an inflammatory fluid), can be sources of pain as well. When ischemic or infarcted, subchondral bone can cause a deep-seated, poorly localized pain. Osteophytes can produce severe, difficult-to-treat radicular pain by compressing nerve roots.

Because OA pain is chronic, psychological factors play an important role and may be related to the individual's perception of pain and psychosocial profile. Pain tends to be more severe in evenings, on weekends, and early in the workweek.[47] Relieving nighttime pain provides the added benefit of allowing restorative sleep.

History and Physical Examination

The patient arrives at the rheumatologist's office using a walker for assistance; she is accompanied by her daughter. Because of pain and instability in her knees, she is unable to mount the examining table without assistance from both her daughter and the physician. The patient reports that for 20 years she has suffered from knee pain, which was originally treated by acetaminophen, later by NSAIDs, and now by a COX-2

inhibitor. She has had recurrent effusions in both knees, and her internist has performed arthrocentesis with joint aspiration and corticosteroid injection 3 to 4 times per year for the past several years. The patient weighs about 20 lb less than she did in her 50s but is still overweight. Over the years, she has received various types of physical therapy and has recently started using a walker because of "instability of her knee," associated with sensations that her knees are periodically "giving way."

On examination, she is 5′3″ tall and 154 lb (BMI, 27). Musculoskeletal examination reveals bilateral bony enlargement of PIP and DIP joints by Heberden's and Bouchard's nodes. She has limited range of motion at both hips but no pain in either site. Her knees are markedly enlarged because of bony hypertrophy, and small to moderately sized joint effusions are present. There is full range of motion in her knees, with marked crepitus.

- **Is there a role for glucocorticoids in OA?**

Orally administered glucocorticoids are not indicated in the treatment of OA. However, intra-articular depocorticosteroids can be useful if there is evidence of inflammation. Synovial effusions should be removed prior to injection. Infiltration of depocorticosteroids also may be helpful in cases of periarticular soft tissue complications, such as anserine bursitis. These agents have not been consistently beneficial in facet joins for treatment of chronic low back pain[48] but have been useful in many patients as epidural injections for symptomatic spinal stenosis.[49]

Despite the belief held by many clinicians that intra-articular depocorticosteroids offer a clear benefit in OA, few published double-blind trials support their benefit over aspiration alone.[50] Some trials have shown short-term benefit.[51] For example, in 1 study, pain relief as assessed by a visual analogue scale lasted for up to 4 weeks when compared with placebo saline injection.[52] No consistent clinical predictors of response to intra-articular depocorticosteroids have been found to aid in patient selection for this therapy.[53] In general, depocorticosteroid injections should be limited to 4 to any single joint per year.[54] Complications of intra-articular depocorticosteroids, such as septic arthritis, are rare if proper aseptic technique is employed.

Depocorticosteroids are crystalline and can induce a transient synovitis or "flare," which occurs within several hours of the injection. In contrast, infection most often develops 24 to 72 hours after the procedure. Application of cold compresses often reduces pain, and the flare resolves within several hours. The suspicion of infection should prompt immediate reaspiration, with subsequent Gram stain and cultures.

- **Are there other intra-articular therapies that provide symptomatic relief?**

Two intra-articular hyaluronic acid (HA) preparations (Synvisc and Hyalgan) are available that can provide symptom relief for several months when given as a series of injections.[55] These drugs are useful in patients in whom simple analgesics and NSAIDs have failed. Synvisc is given in a series of 3 injections 1 week apart; 5 injections are given for Hyalgan. These HA derivatives can reduce pain for several months, as stated above, and may also improve mobility. Their mechanism of action is unknown. Possible mechanisms include an anti-inflammatory effect, a short-term lubricant effect, an analgesic effect by directly buffering synovial nerve endings, and a stimulating effect on synovial lining cells, causing them to produce endogenous HA.

- **What is the role of physical therapy in OA management?**

Physical measures make up an important part of any successful therapeutic program for OA. A variety of physical modalities can be used to relieve pain, reduce stiffness, and limit muscle spasm while strengthening periarticular structures to provide improved joint support. Physical measures in OA management include exercise, supportive devices, alterations in activities of daily living, and thermal modalities (**Table 3**).

The most commonly used physical measure is exercise. Exercise programs have been linked to reduced pain and improved function in patients with OA. In most cases, patients can perform either range of motion or active strengthening exercises after minimal instruction. Such exercises help to improve muscle tone, which reduces muscle spasm and prevents contractures.[56] Improved strength of periarticular structures resulting from exercise adds stability and support to the joint, appears to reduce symptoms, and may retard disease progression. In patients with OA of the knee, strengthening of the quadriceps muscles can improve function and decrease pain for up to 8 months.[57] Currently used aerobic exercises include walking,[58] swimming, and stationary bicycle riding. Swimming is useful in nearly all forms of OA except lumbar facet OA, in which symptoms may be worsened by hyperextension of the spine. The onset of any exercise program should be carefully graded; if the regimen is advanced too quickly, symptoms may initially worsen and the patient may not comply with the exercise program.

Among specific exercises sometimes used in back pain are the Williams flexion exercises, which involve flexion of the neck, trunk, pelvis, and legs. These exercises are indicated for musculoskeletal low back pain and mild spinal stenosis and are contraindicated in patients with osteoporosis or symptomatic herniated nucleus pulposus. In patients with the latter disorder, McKenzie extension exercises could be used.[59] There is no advantage to bed rest for patients with either acute or chronic low back pain; these patients should resume and/or maintain their daily activities as much as possible.[60]

Supportive devices are often of value as a supplement to the exercise program by partially unloading the joint with activity. These devices include canes, crutches, walkers, corsets, collars, and shoe orthotics. Canes can decrease loading on joints in the lower body by up to 60%.[61] The length of a cane should be equal to the distance between the upper border of the femur's greater trochanter and the bottom of the shoe heel; the cane should be held in the hand contralateral to and moved together with the affected limb. Knee braces are also an option in some patients with tibiofemoral disease, especially those with lateral instability.

Thermal modalities can be particularly effective. There is no advantage of superficial heat modalities over cold applications; thus, the use of heat, cold, or alternating heat and cold is based on patient preference. Traditionally, the more acute the process, the more likely cold applications will provide benefit.[62]

There are several miscellaneous physical modalities, such as massage, yoga, acupressure, acupuncture, pulsed electromagnetic fields, transcutaneous neural stimulation, and spa therapy. However, these modalities have not been prospectively validated for OA pain management.

Treatment and Follow-up

The rheumatologist decides to try a hyaluronic acid preparation along with formal referral to physical therapy for quadriceps strengthening and gait training. The COX-2 inhibitor is continued. The patient achieves a weight loss of 10 lb over the following 6 months and reports symptomatic improvement at follow-up visits.

CASE STUDY 3
Initial Presentation

A 65-year-old man with long-standing symptomatic OA of the hips, lumbar spine, and knees goes to an emergency department (ED) because of a 1-week history of worsened pain in his right knee, associated with increased swelling. He has not had any fever or chills or

Table 3. Physical Measures in OA Management

Exercise

Active: range of motion, isometric, isotonic, isokinetic

Passive range of motion

Rest periods

Support devices and orthotics

Canes, crutches

Collars

Medial taping of patella, knee braces

Shoe insoles

Modified activities

Adjusting furnishings (eg, height of toilet seat)

Proper posture when sitting, sleeping, or driving

Thermal modalities

Cold application (eg, cold packs, vapocoolant spray)

Deep heat (eg, ultrasound)

Superficial heat (eg, hot pack, paraffin baths)

Miscellaneous

Acupuncture

Chiropractic

Pulse electromagnetic fields

Spa, massage, yoga

TENS

OA = osteoarthritis; TENS = transcutaneous electrical nerve stimulation.

recent trauma to the joint but is concerned because the pain seems worse than it has been previously.

History

The patient has been taking a COX-2 inhibitor for pain relief because of a history of peptic ulcer disease. He also has a history of bilateral knee effusions, which have been periodically drained, and has received corticosteroid injections to his knee joints. With each injection, pain relief has lasted weeks to months. The patient's last arthrocentesis was 3 months ago.

Physical and Radiologic Examinations

On examination, the patient is afebrile. Bilateral knee bony enlargement and deformity are observed, along with marked crepitus. The right knee is warm, with a moderately sized effusion. A radiograph taken in the ED confirms severe OA of the right knee with marked medial compartment narrowing, subchondral sclerosis, and large osteophytes. The ED physician suggests doubling

the patient's dose of COX-2 inhibitor for a few days to reduce the inflammation and pain associated with his arthritis and following up with his internist if the pain does not improve.

Follow-up with Internist

Three days later, the patient goes to his internist for a follow-up examination; he is now limping, with an antalgic gait, and reports worsening right knee pain. He again reports no fever or systemic symptoms. Examination reveals a temperature of 100.4°F, pulse of 91 bpm, blood pressure of 113/65 mm Hg, and respiratory rate of 18 breaths/min. The right knee is erythematous and warm and has a large effusion; the patient is unable to extend his knee past 120° without marked pain. His internist decides to perform arthrocentesis, from which 40 mL of purulent fluid is aspirated. Leukocyte count is $140 \times 10^3/mm^3$ (90% polymorphonuclear cells). The patient is started on intravenous oxacillin. Twenty-four hours later, culture from the aspirated fluid grows gram-positive cocci in clusters, which are determined to be methicillin-sensitive *Staphylococcus aureus.*

- **Should a diagnostic arthrocentesis have been performed in the ED?**

In this case, the ED physician was misled by the patient's history of severe OA and by the radiograph that confirmed the presence of advanced OA. Pre-existing joint disease of any cause, including OA, predisposes to septic arthritis. Even in this patient with an established diagnosis of OA, arthrocentesis should have been performed promptly to investigate the cause of effusion and pain. The differential diagnosis for such acute exacerbation of knee pain should have included not only infection but also the crystal arthropathies, particularly calcium pyrophosphate deposition disease. Radiographs obtained early in the course of septic arthritis may show only subtle signs or may appear completely normal and thus are not helpful in ruling out septic arthritis. The fact that this patient was afebrile on presentation is unusual; however, fever is not a universal feature of monarticular joint infection. Also, the patient was on a standing dose of an NSAID, which may have diminished his febrile response.

Additional Follow-up

The patient receives antibiotics, intravenously, and arthroscopic joint lavage and recovers from the acute infection. One year later, however, he reports more pain in his right knee. Follow-up radiographs demonstrate much more extensive disease in that knee.

- **What other diagnoses should be considered in a patient with knee pain and OA?**

Other common causes of "knee" pain that are actually extra-articular in origin are frequently seen in patients with OA. Although anserine bursitis and prepatellar bursitis should be relatively easy to distinguish from actual arthritis, these diagnoses are often missed.

CONCLUSION

The main symptom of OA is pain. Many potential sources for OA pain exist, and a correct diagnosis can lead to more effective medical management. Medical therapy of OA often combines nonpharmacologic approaches (eg, temperature modalities, exercise, weight loss) with appropriate intra-articular, topical, and/or systemic pharmacologic therapy. Treatment must be individualized and take into account risk factors (eg, age, comorbidities, prior history of peptic ulcer disease) that can lead to adverse medication-related events. **HP**

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