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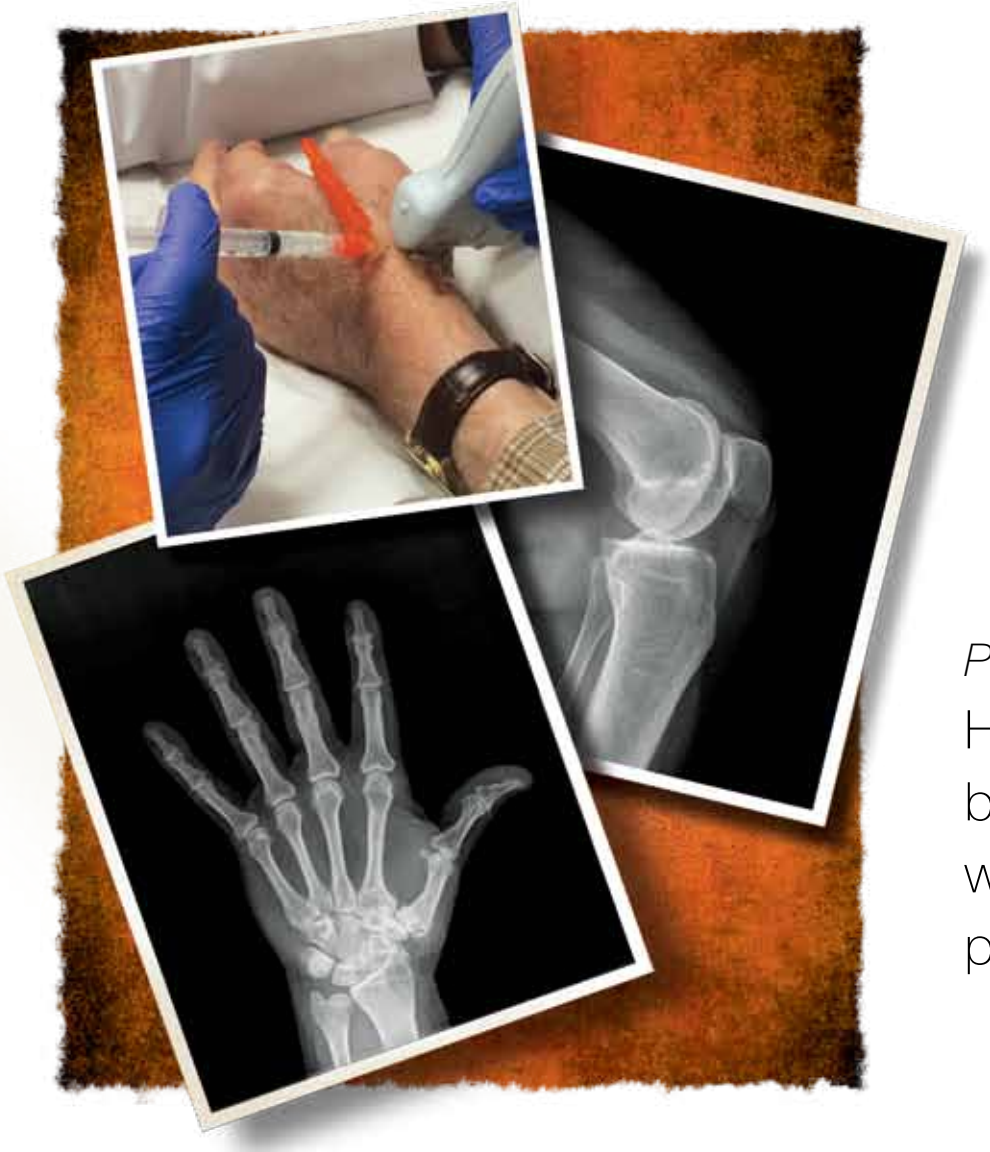
Osteoarthritis pain: An integrated approach



PLUS:

How to set boundaries with chronic pain patients

March 2014

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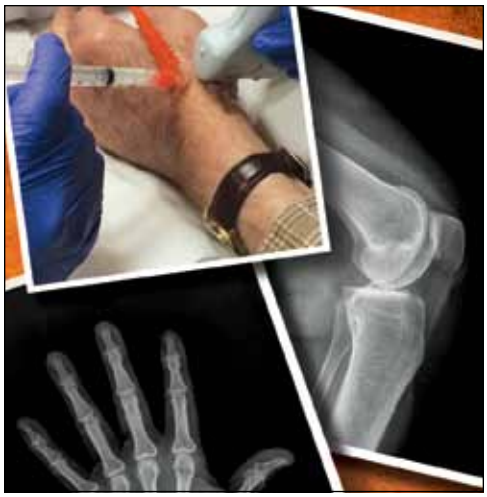

S9

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How to set boundaries with chronic pain patients

While a collaborative relationship is optimal for pain management, there may be times when saying *No* is the best treatment. This review—and easy-to-use "decision tree"—can help with both.

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Joe C, age 55, an African American patient whose care you are monitoring for a colleague on leave, is being treated for low back pain that he attributes to an injury sustained in the military. This is his second appointment with you.

On his first visit—a month after your colleague started Mr. C on an opioid trial (hydrocodone/acetaminophen 5 mg/325 mg every 6 hours as needed for pain)—you titrated the dose to 10 mg/325 mg because he reported inadequate pain relief. Now the patient says the pain is worse than ever. He denies experiencing any opioid-related adverse effects and requests a higher dose. Yet there is no indication that the opioid has been helpful. What's more, when you ask whether he has begun an exercise program or implemented any of the other self-management strategies discussed at his previous visit, the patient shakes his head "No."

What's the next step?

Illustration: Steve Dininno

Disclosure
The author reported no potential conflict of interest relevant to this article.

Effective pain management has been deemed a human right,[1] but some chronic pain patients perceive that to mean they are entitled to opioid analgesics for prolonged pain control.[2] In response to these expectations, clinicians may feel pressured to continue prescribing opioids—thereby reinforcing the patient's beliefs and reliance on medication.[3] This has contributed to a dramatic rise in opioid analgesic misuse and deaths from prescription drug overdose,[4] identified by the Centers for Disease Control and Prevention as a "public health epidemic."[5]

Clinician and patient share responsibilities

The right to effective pain management comes with responsibilities that patient and clinician share[6]—a model of collaborative care also known as a "working alliance." The benefits of such an alliance, a concept that originated in the mental health arena and is especially important in the realm of pain management, has been validated by strong research support.[7] Patients who have rewarding relationships with their providers have better outcomes[8] and are less likely to seek assistance from other sources,[9] which reduces the risk of conflicting treatment plans and further confusion.

Yet chronic pain poses numerous challenges to a healthy patient-provider relationship, with problems such as power struggles, distrust, and feelings of stigmatization increasing with the duration of illness.[10,11] Research has shown that some patients perceive their providers as lacking in empathy, doubting that their pain is real, and being influenced by stereotypes. This is in contrast to the perceptions of clinicians, some of whom have acknowledged being more concerned about other urgent health conditions than about chronic pain, looking for objectivity within what is largely a subjective condition, and not taking ample time to build a relationship of trust.[10] Such issues are critical in pain management, as the success of the patient-provider collaboration often determines whether a patient will adopt self-management strategies.[3]

Establishing boundaries

Increased emphasis on communication has been proposed as a way to improve the patient-provider relationship,[9] and communication training for providers has been shown to be beneficial.[12] Essential elements of a healthy relationship include compassion, clear expectations (setting boundaries), and adequate explanations on the provider side, and active participation and involvement in decision making on the part of the patient.[12]

Pain management, in particular, requires appropriate boundaries. This is crucial regardless of the treatment plan, in part because clinicians often find it hard to identify potential "ruptures" in their relationships with patients.[6,13] Boundaries are simply rules or limits that individuals create to identify reasonable, safe, and permissible ways for others to behave around them—and to determine how they'll respond when someone oversteps these boundaries.[14] (To evaluate your boundary-setting skills, take the self-assessment quiz in the TABLE.[15])

Difficulty setting boundaries? Establishing appropriate boundaries is a skill that requires a lot of thought and practice, yet many clinicians learned little about it in medical school or clinical training. To master this skill, it is important to recognize that a boundary is not a threat or an attempt to control the

TABLE
Setting boundaries: Assess your abilities[15]

A "Yes" to any of the following indicates difficulty in setting boundaries:
☐ Do you have trouble saying "Yes/No" or find it difficult to accept "Yes/No" from others?
☐ Do you tend to take on, or experience, others' pain or problems?
☐ Do you share too much personal information—or share no personal information at all?
☐ Are you unable to express your needs, wants, and reactions or to ask for help?

behavior of others—and that setting appropriate limits will ultimately improve, rather than detract from, relationships with patients.

There are 4 steps involved in setting appropriate boundaries:[15]

1. **Name** or describe the behavior that is unacceptable.
2. **Express** what you need or expect from the other person.
3. **Decide** what you will do if he or she does not respect the boundaries that you have established.
4. **Validate** your actions by recognizing that setting boundaries is important work and that your rights are important.

CASE

Before determining a course of action for Mr. C, you conduct a pain assessment to rule out new pathology. Finding nothing, you tell the patient you're concerned because you have already made changes to his pain medication and discussed other things he can do to feel better. You go on to explain that despite the widely held perception that opioids are the most potent medications available for the treatment of pain, there is little evidence that they are more effective than other therapies.[16]

You tell him that you can't continue to increase his medications without incorporating some other strategies and remind him that a lot of chronic pain management involves self-management strategies.

Before you have an opportunity to elaborate, Mr. C objects. Patients usually anticipate that a treatment plan will provide fast relief and not require significant lifestyle changes.

Now what?

Need additional help? Use the pain management "decision tree"

Although there are complex guidelines for the management of opioid therapy, simplified decision support tools to guide difficult discussions and assist in determining a course of treatment for chronic pain patients are scarce. What's needed is an easy-to-use pain management "decision tree," like the adaptation on page S6 (FIGURE).[17]

It starts with the first step in pain management—having a new or established patient with a chief complaint of pain. The next step is a comprehensive pain assessment. This should include a psychological evaluation with an assessment of the risk of addictive disorders, an appraisal of pain level and function, and a diagnosis with the appropriate differential.[18]

Once a decision is made to work with the patient, the provider must determine whether the pain is acute or chronic and educate the patient about the difference. (Acute pain has a sudden onset, lasts no more than 6 months, and resolves when the underlying cause is treated, while chronic pain persists beyond the "normal" healing time—even if it originated from a trauma, injury, or infection—and is affected by both physical symptoms and emotional problems.[18])

Outline treatment goals and review options

For a patient with chronic pain, it is helpful (and promotes a collaborative relationship) to outline treatment goals and consider an array of evidence-based therapies that include nonopioid medications, physical therapy, behavioral programs such as cognitive-behavioral therapy, and procedures such as a nerve block.[19]

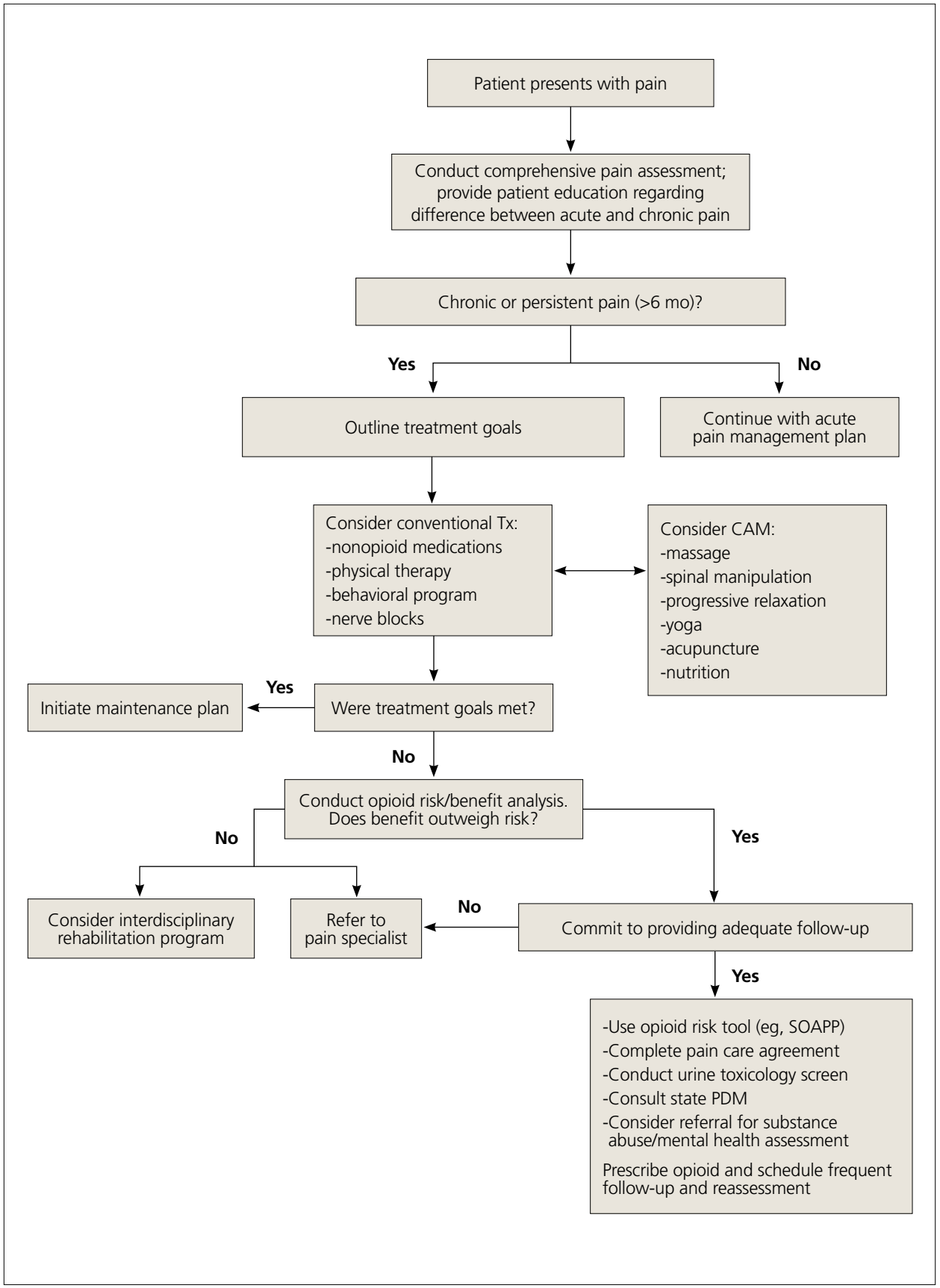
Review alternative modalities. Empirically validated complementary and alternative therapies such as spinal manipulation, massage, yoga, and acupuncture can be considered at this time, as well.[20] This presents an opportunity to educate the patient about the range of nonpharmacologic pain management strategies, attempt to integrate patient preferences, and encourage joint decision making. It may be helpful, too, to expand the conversation to include treatment outcomes that focus not solely on the reduction or control of pain but on effective functioning within the context of continued pain.[8]

When to consider opioids

Only after other treatment options have been exhausted should an opioid trial be considered.[21] As the decision tree shows, a careful risk-benefit analysis is required. Routine assessment of analgesia, activity, adverse effects, aberrant behavior, and affect will help to direct therapy.[11] If it is determined that the risk outweighs the benefit, a referral to a pain specialist or an interdisciplinary rehabilitation program is indicated. If, on the other hand, the benefits outweigh the risks, it is crucial to ensure that the clinician's practice will be able to provide adequate

Only after other treatment options have been exhausted should an opioid trial be considered.

FIGURE: A decision support tool for chronic pain management[17]



CAM, complementary and alternative medicine; PDM, prescription drug monitoring database; SOAPP, Screener and Opioid Assessment for Patients with Pain.

patient support: visits every week to month for a high-risk patient on opioids, and once every 3 months for a more stable patient.[21]

A risk assessment tool *(eg, Screener and Opioid Assessment for Patients with Pain [SOAPP]*, available at www.painedu.org/soapp.asp) can be used to determine the extent of monitoring required based on the patient's relative risk for developing problems when placed on long-term opioid therapy.[22]

A pain care agreement is also recommended at this time. It helps patients understand the clinician's expectations, the short-term nature of opioid therapy, the risks that may be incurred, and the way various scenarios will be handled. For example, a pain care agreement might outline how lost or stolen opioids, requests for early refills, noncompliance with scheduled appointments, and other aberrant behaviors (eg, diversion, doctor/pharmacy shopping, violence, and threats) will be handled.

A urine toxicology screen should be obtained, as well. If a patient tests positive for an illicit substance (eg, cannabis, cocaine, or heroin), a face-to-face discussion outlining the conditions that must be met in order to initiate or continue opioid therapy (Step 2 of boundary setting) is crucial. It may be useful, too, to consult the prescription drug monitoring electronic database, which collects designated data on controlled substances dispensed within participating states. Prescription drug monitoring helps providers educate their patients about the use, abuse, diversion of, and addiction to prescription drugs; legitimize the medical use of controlled substances for their patients; and facilitate and encourage the treatment of prescription drug addiction.

After collecting all this information on the patient, a provider may decide on a referral to substance abuse and/or mental health services to complement current treatment, despite the patient's reservations. A professional and tactful approach will help ensure patient safety and compliance in such cases. And, for a clinician who will be continuing to treat the patient, this is the time to implement Steps 2 and 3 in boundary setting: Clearly state what behavior is expected and what the consequences will be if those expectations are not met.

CASE

In response to Mr. C's resistance, you review the pain care agreement completed during his last visit and ask him to complete the SOAPP. You also request a urine specimen for a toxicology screen. He denies these requests, angrily stating, "I did that already, and I don't need to do it again," and continues to push for a dose escalation. You remind him that you, too, are concerned about his pain, but that modern medicine does not offer a quick fix. He finally agrees to complete the SOAPP and have the necessary lab tests when he realizes he will not get his prescription renewed otherwise.

When you review the results of these tests, you realize a higher level of monitoring is not needed. According to the SOAPP results, Mr. C appears to be compliant with his opioid regimen, and the urine screen indicates that he is not using any illicit substances. You decide that a continuation of the opioid trial is warranted.

You switch Mr. C to a trial of long-acting morphine (15 mg every 8 hours), with short-acting morphine 15 mg twice a day as needed; request that Mr. C come in at least once a month for the next several months; and refer him to physical therapy to move him toward a self-management approach.

Before the patient leaves, you implement Step 3 of boundary setting: You make it clear that if he fails to show up for his scheduled appointments or to go for the physical therapy consult, you will institute a taper plan that will end with the termination of the opioid trial.

Boundary setting is not always comfortable

Many clinicians feel uncomfortable during the boundary-setting process. This is where Step 4 comes in: remembering that setting boundaries is important work because your rights as a provider are important. When reasonable limits are placed on a patient and the patient continues to step beyond those limits, it is imperative that you maintain your boundaries and be consistent in your message. Sometimes, saying "No" is the appropriate treatment.

A boundary is not a threat or an attempt to control the behavior of others. Setting appropriate limits will improve—rather than detract from—your relationships with patients.

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FEATURE ARTICLE

An integrated approach to osteoarthritis pain

Informed therapeutic choices can help patients with osteoarthritis pain in 2 common locations—the hand and the knee—stay active.

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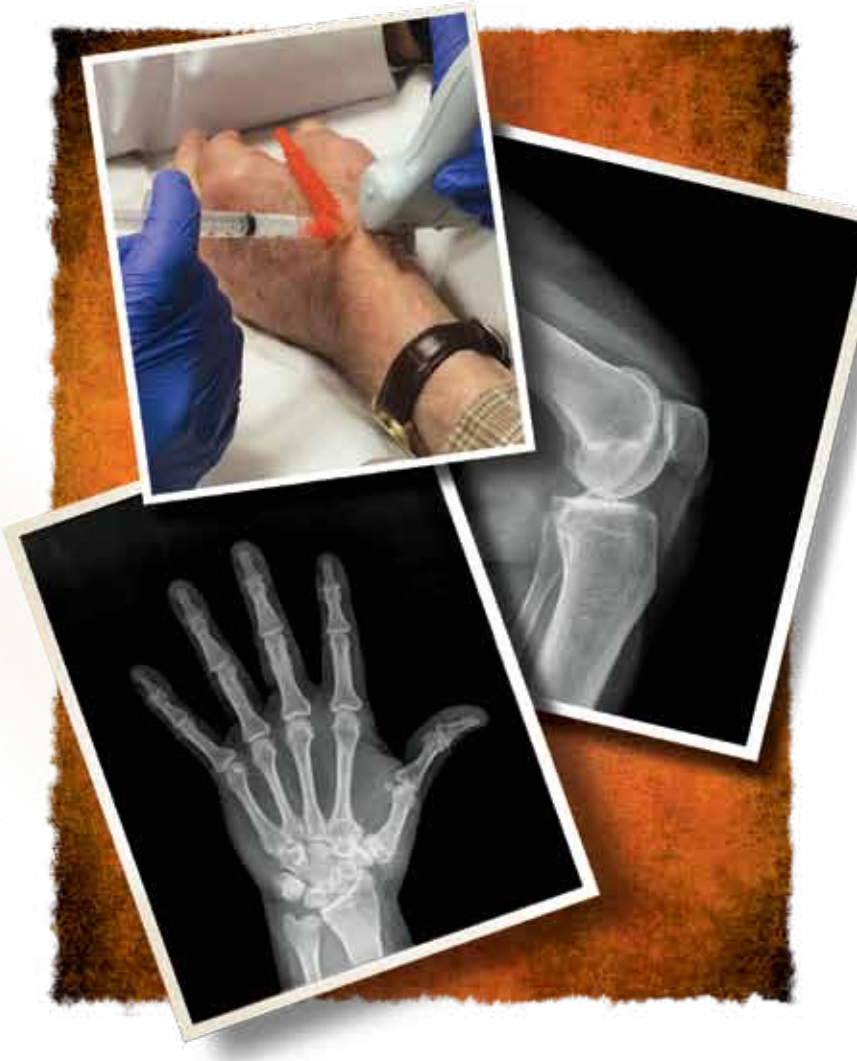
Osteoarthritis (OA) is not a normal consequence of aging but a degenerative joint disease caused by trauma, mechanical forces, genetics, and inflammatory factors.[1] OA affects nearly twice as many women as men over age 60 (18% vs 9.6%, respectively).[2] The most common sites are the hands, knees, hips, spine, and any joint that has sustained traumatic injury. Joint pain is the principal symptom, but swelling, deformity, stiffness, and loss of function also occur.

In primary care, it's quite common to see OA in 2 specific sites—the thumb's first carpometacarpal (CMC) joint and the knee. This article describes a patient with OA affecting both sites. We review the workup and diagnosis of these presentations and offer an integrated approach to managing OA symptoms.

CASE

Mr. D, age 70, has a 4-year history of pain in his left knee and at the base of his left thumb in the CMC joint. His thumb is particularly sore today, he says, and this impedes his work as a commercial artist. He scheduled this appointment for repeat glucocorticoid injections, which usually help him for about 3 months before the pain recurs.

Mr. D walks with a cane, and his symptoms worsen with cold weather. His painful joints show no mechanical signs or symptoms (such as "giving way"), and he reports no history of falls or trauma. To control his pain, Mr. D applies topical diclofenac 1% gel twice daily and occasionally uses oral hydrocodone/acetaminophen, 5 mg/300 mg. The hydroco-



Radiographs and photo courtesy of Suraj Achar, MD, FAAFP
Photo composition: John J. DeNapoli

Disclosure
The authors reported no potential conflict of interest relevant to this article.

done tends to cause constipation, which he manages with docusate sodium and senna.

An occupational therapist created a thermoplastic thumb splint for him and performed iontophoresis with dexamethasone, which was temporarily effective. Mr. D does not follow through with stationary bicycle exercise recommendations. Orthopedic surgeons have offered surgical options for his thumb and knee, but Mr. D has wanted to delay surgery.

Osteoarthritis of the hand

OA can affect multiple joints in the hand, particularly the proximal and distal interphalangeal joints. Metacarpophalangeal (MCP) joints—as well as those in the carpal rows—can be affected, but clinically far less often than the first CMC joint. Because the thumb is more prone to injury than the other fingers, it is more likely to develop degenerative changes.

OA in the first CMC joint, also known as the basal or trapeziometacarpal joint, can cause considerable pain and functional limitation. All of the thumb's 5 primary movements—abduction, adduction, extension, flexion, and opposition—are critical for grasping and pinching. CMC arthritis affects more women than men,[3] perhaps because of gender-related hormonal or anatomic differences.

The "grind test" can help you make the diagnosis

Clinically, OA at the first CMC joint presents with diffuse pain that worsens with certain movements. Some individuals also complain of weakness. Pain typically is exacerbated by simple tasks—such as opening jars, turning keys, sewing (pinching), and gripping—and usually is relieved with rest.

A deformity caused by synovial hypertrophy, osteophytes, and subluxation may exist at the base of the metacarpal, and crepitus can be palpated at the joint. Mild tenderness may be present over the volar plate, and pinch strength frequently is reduced. Rheumatoid arthritis at this joint is differentiated from OA by tendon laxity, joint effusions, and swan neck deformity.

The "grind test" is a useful way to confirm OA of the CMC. The first metacarpal is held firmly while providing an axial load into the joint and rotating the base of the metacarpal in different directions (see the illustration, below left). A positive grind test will reproduce the patient's symptoms and palpable crepitus in the joint. A negative test, however, does not necessarily correlate with a lack of radiographic evidence of OA.[4]

Imaging. Plain films with dorsal palmar (FIGURES 1, 2) and oblique views usually suffice in making a diagnosis of OA of the hand. Lateral views may be more difficult to interpret because of overlapping bones.

Ultrasonography can define degenerative changes and swelling as well as provide dynamic imaging.[5] Mild joint space narrowing, subchondral sclerosis, mild laxity, and effusion with no significant subluxation mark early stage disease of the first CMC joint. Bone spurring and loss of joint space can be seen in later stages. Imaging of the hand may also show erosive or osteoarthritic changes in the distal and proximal interphalangeal joints, commonly known as Heberden's and Bouchard's nodes.

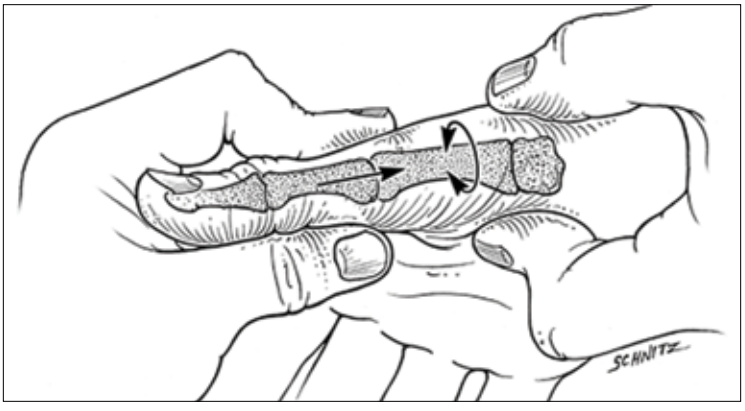
Treatment begins conservatively, with PT and NSAIDs

Conservative management for pain and functional limitation of OA of the CMC consists of 3 to 4 weeks of activity modification, joint protection, muscular strengthening (physical therapy), nonsteroidal anti-inflammatory drugs (NSAIDs), and splinting. Guidelines recommend topical NSAIDs before oral therapies for OA.[6]

Topical diclofenac 1% gel has shown efficacy in hand OA. It can be applied 4 times daily at 2 g/application. Reported adverse effects include local rash, itching, and burning, but no increase in gastrointestinal (GI) events.[7,8] Cap-

HOW TO PERFORM THE "GRIND TEST"

Hold the first metacarpal firmly while providing an axial load into the joint and rotating the base of the metacarpal in different directions. A positive test will reproduce the patient's symptoms of pain as well as palpable crepitus in the joint.



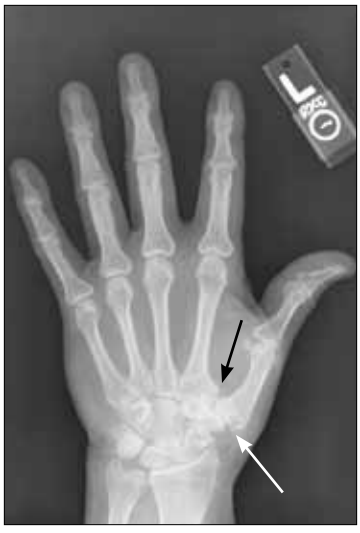
Reprinted, with permission from: Arthritis at the base of the thumb. Indiana Hand to Shoulder Center Web site. Available at: www.indianahandtoshoulder.com/medical_education_hand_arthritis.html. Accessed January 24, 2014.

saicin also has shown efficacy for OA in randomized trials,[9] although its cost, frequency of application, local irritation, and less robust evidence of efficacy limit its usefulness.[6]

Oral NSAIDs can be used for a short period or as needed, but long-term use is not recommended because of the risk of GI, renal, and cardiac complications.

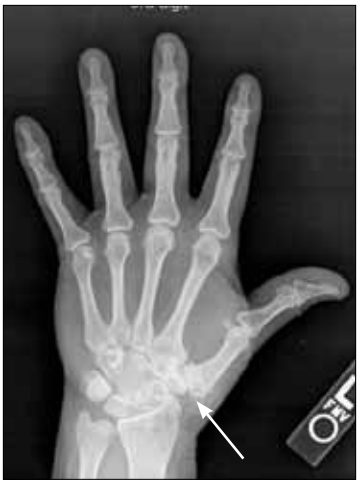
Acetaminophen, like oral NSAIDs, can be used as needed. Caution patients to limit total acetaminophen use to no more than 8 500-mg tablets (4 g) in any 24-hour period. To avoid unintentional overdose, monitor patients taking daily acetaminophen, especially those with liver disease or who consume 3 or more alcoholic drinks per day.[10] Avoid opioids, especially for patients over age 65 because of increased risk of adverse effects, particularly sedation, confusion, and constipation.

FIGURE 1



Radiograph (anteroposterior view) of patient with severe osteoarthritis (OA) of the first carpometacarpal (CMC) joint with subluxation (white arrow), along with mild-to-moderate OA of the tri-scaphoid and second CMC joints (black arrow).

FIGURE 2



Same patient 1 year later (anteroposterior view) shows severe degenerative joint disease of the first CMC joint, with sclerosis and cystic changes (arrow).

Pain continues? Consider a glucocorticoid injection

When conservative treatment fails to adequately control pain, joint injection can be performed with a combination of a long-acting anesthetic and a glucocorticoid.[3] Although some studies have shown no long-term pain relief with joint injections, others have shown the procedure is well-tolerated, improves function, and can provide pain relief—especially in early OA.[11,12] Radiographic guidance with ultrasound (see photo inset, page S9) or fluoroscopy can help with injection placement and improve outcomes.[13,14] A short burst (10 to 15 seconds) of ethyl chloride spray, a rapidly evaporating coolant, may be beneficial prior to the glucocorticoid injection to decrease the pain of the injection.

Several randomized controlled trials and meta-analyses of intra-articular glucocorticoids have demonstrated short-term benefit without harm.[11,15] Agents shown to be helpful include methylprednisolone, triamcinolone acetonide and hexacetonide, betamethasone, and dexamethasone (TABLE 1). Because dexamethasone sodium phosphate has both rapid onset and short action, it is often combined as a bridge agent with a longer-acting glucocorticoid such as methylprednisolone, triamcinolone, or betamethasone.[16] Intermediate-acting agents also can be used as monotherapy. The shorter acting the glucocorticoid, the less likely it is to cause a post-injection flare. The less soluble the agent, the longer the effect.

Anesthetic agents added to the injection provide early pain relief and confirm placement. Available local anesthetics include long-acting bupivacaine 0.25% (8 hours) and short-acting lidocaine 1% and 2% (1 hour).[16,17] In our experience, small joints (hand, sternoclavicular, and acromioclavicular joints) usually require 0.25 to 1.0 mL of anesthetic, whereas medium joints (elbow and wrist joints) can use 1 to 2 mL, and large joints (knee, ankle, shoulder, hip) can require from 3 to 5 mL.

3 surgical procedures worth considering

If pain and functional limitations are refractory to conservative measures, surgical evaluation is warranted. Two surgical procedures—trapeziectomy with hematoma distraction arthroplasty and hemitrapeziectomy with osteochondral allograft—are effective in relieving pain of severe first CMC joint arthritis.[18] Another common procedure is ligament reconstruction with tendon interposition (LRTI). These 3 procedures involve complete or partial excision of trapezium and filling with a spacer such as the flexor carpi radialis tendon, abductor pollicis longus tendon, palmaris longus tendon, a silicon rubber block, or a joint prosthesis. Postoperative disability and pinch strength are similar among these 3 procedures.[18] After surgery, patients undergo 5 to 18 weeks of splinting and physiotherapy to restore motion and strength.

A 2011 systematic evidence review found no surgical procedure to be superior to another for OA of the thumb CMC. Long-term benefits could not be assessed because follow-up studies were relatively brief (12 months). The authors also concluded[19]:

- When interposition is performed with trapeziectomy, autologous tissue interposition appears to be preferable.
- Trapeziectomy with LRTI seems associated with higher complication rates.

Osteoarthritis of the knee

Symptomatic OA of the knee affects 10% to 20% of adults age 55 or older.[20] Age is the strongest risk factor for developing OA. Independent predictors of radiographic knee OA include female gender, overweight (body mass index >25.9), localized knee pain, previous injury, restricted range of motion in flexion, crepitus, and effusion.[21]

Morning stiffness, crepitus among criteria for knee OA diagnosis

OA of the knee presents as unilateral or bilateral pain with insidious onset over weeks, months, or years. Pain is worsened by activity and relieved by rest. Patients may report morning stiffness, usually lasting <30 minutes. Knee pain may be exacerbated by activities such as walking down stairs or carrying heavy objects. Joints may become swollen after activity.

Physical examination. Palpation may reveal tenderness of the affected joint line or patellofemoral joint, edema of the joint, and crepitus. In severe disease, bony osteophytes may be felt. A genu varus or valgus deformity may develop as OA progresses to affect the lateral and medial knee compartments.[22] Physical findings may underestimate disease severity.[23]

Clinical diagnosis of knee OA can be made (with 95% sensitivity and 69% specificity) in a patient meeting 3 or more of these criteria[24]:

- age >50 years
- morning stiffness that lasts <30 minutes
- crepitus on exam
- bony tenderness to palpation
- enlargement of the bone
- no warmth of the joint.

Lab testing for erythrocyte sedimentation rate and rheumatoid factor, aspiration of synovial joint fluid, and radiographs of the affected knee(s) may be indicated to rule out other infectious or inflammatory causes.

Imaging. Plain radiographs are usually used to assess knee OA presence and severity (FIGURES 3, 4). Positive findings include joint space narrowing, subchondral sclerosis, osteophytes, subchondral cysts, and subluxation of the joints.[20] Significant radiographic changes

TABLE 1
Intra-articular glucocorticoids for OA: Dosing and duration

Corticosteroid	Concentration (mg/mL)	Steroid equivalent (mg)	Onset/ duration	Amount (mL) based on joint size		
				Small	Medium	Large
Dexamethasone sodium phosphate	4	8	S	0.1	0.1 - 0.25	0.25
Methylprednisolone acetate	40	40	I	0.25	0.5 - 1.0	1.0 - 2.0
Triamcinolone acetonide	40	40	I	0.25 - 0.5	0.5 - 1.0	1.0 - 2.0
Triamcinolone hexacetonide	20	40	L	0.25	0.25 - 0.5	0.5 - 1.0
Betamethasone sodium phosphate and betamethasone acetate	6	8	L	0.25 - 0.5	0.5 - 1.0	1.0 - 2.0

I, intermediate; L, long; OA, osteoarthritis; S, short.

Source: DailyMed Web site. US National Library of Medicine, National Institutes of Health, Health & Human Services. Available at: www.dailymed.nlm.nih.gov/dailymed. Accessed January 25, 2014.

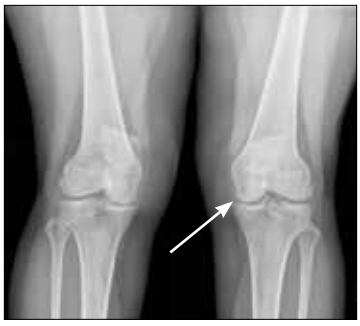
may not be seen in early OA. Because cartilage is aneural, some patients may have minimal complaints despite moderate to severe abnormalities on imaging. One study found that[25]:

- only 50% of patients with signs of OA on radiographs were symptomatic
- only 50% of symptomatic patients showed changes on radiographs.

Even so, plain films can be a useful tool for determining degree of radiographic disease and monitoring progression when patients show significant worsening of symptoms.

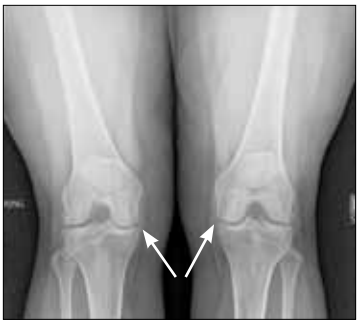
When radiographic OA is present, magnetic resonance imaging (MRI) is rarely indicated. In early OA, MRI can be used to evaluate for partial- or full-thickness cartilage defects and bone marrow edema (best noted by increased signal in T2 or other fluid sensitive sequences in the subchondral bone).[26] Subjective knee pain does not reliably correlate with MRI findings of OA.[27]

FIGURE 3



Radiograph of a 67-year-old woman with knee pain. Anteroposterior view shows bilateral OA predominately in the medial femoral tibial compartments (arrow), manifested by joint space narrowing and osteophyte formation. There is no knee effusion or focal soft tissue swelling. No intra-articular bodies are seen.

FIGURE 4



Same patient, weight-bearing Rosenberg view of the knees. Bilateral medial compartment narrowing (arrows) is more apparent in the Rosenberg view, taken standing with the knees bent at 30 degrees.

Conservative treatment options include OT, PT, and diet modification

Primary care therapy for OA of the knee has 3 goals: decrease pain, limit disability or decrease in function, and educate patients about the course of the disease. It is important to inform patients that although OA is a chronic, progressive disease, most people can manage their pain and functional limitations by adhering to the available therapeutic options.

Patients whose treatment includes arthritis education or self-help classes, such as those offered by the Arthritis Foundation (www.arthritis.org), report decreased pain and improved quality of life even 4 years after the initial diagnosis. They also require significantly fewer physician visits.[28]

Occupational therapy. A simple slip-on neoprene brace worn 7 hours daily for 6 weeks can reduce pain and bone marrow edema by 25%.[29] Valgus bracing of the knee offloads the medial compartment in patients with predominant medial-sided knee OA. When used regularly, these braces can improve function and pain, but their cost and bulk may limit their usefulness.[30] Some patients benefit from iontophoresis, with dexamethasone administered transdermally to allow localized delivery with decreased systemic effects.

Physical therapy, exercise, and diet. A therapeutic exercise program can improve mobility, increase lower extremity strength and daily function, and decrease pain.[28] Exercise is particularly effective for improving symptoms of OA of the knee and hip. The most effective regimen consists of exercises to increase strength, flexibility, and aerobic capacity.[31]

Obesity, hyperglycemia, and other metabolic issues have been shown to accelerate OA progression. (See "Obesity-related pain: Time for a new approach that targets systemic inflammation," from *Chronic Pain Perspectives*. 2013;62(9):S22-S28. Available at: www.chronicpainperspectives.com/articles/feature-article/article/obesity-related-pain-time-for-a-new-approach-that-targets-systemic-inflammation/1c084996f70d3c940e4f77d4e704a4b9.html.) Combining exercise with dietary interventions can slow OA progression while improving pain, function, and quality of life.[32]

Oral therapy. Although acetaminophen and NSAIDs have shown similar efficacy for knee OA, acetaminophen is the preferred first-line agent, according to the American College of Rheumatology (ACR).[33] NSAIDs are associated with higher rates of GI bleeding, peptic ulcer disease, and renal failure.[33,34] Because of the risk of acute liver toxicity with acetaminophen overdose, caution patients not to exceed the recommended maximum of 4 g/day.[33]

If maximum-dose acetaminophen alone does not adequately improve symptoms, concurrent oral or topical NSAIDs may be added.[33] Because all NSAIDs have comparable efficacy, choose those with the lowest risk of GI bleeding, such as ibuprofen or diclofenac.[34] Educate patients about the risks and benefits of NSAID therapy.

International guidelines[35,36] rarely call for long-term opioid therapy for knee OA except

TABLE 2
Adjunctive pharmacologic options for OA management

Agent	Proposed benefit	Risk	Evidence
Glucosamine/chondroitin supplements	Potential pain improvement	Generally safe and well tolerated	GAIT, a multicenter RCT sponsored by NIH, found glucosamine alone did not reduce pain in patients with OA; patients with moderate-to-severe OA may experience some improvement with combination glucosamine/chondroitin as an adjunct therapy[39,40]
S-Adenosylmethionine (SAMe) supplements	Symptomatic improvement in pain and functionality	Tolerability similar to placebo and better than NSAIDs	Meta-analysis of 11 RCTs found SAMe improved OA pain and increased function at a rate comparable to NSAIDs, with fewer adverse effects[41,42]
Colchicine	Decreased frequency and intensity of OA attacks	GI upset/bleeding, gout	In RCTs, patients receiving adjunctive colchicine twice daily had greater symptomatic benefit at 12 and 20 weeks, compared with placebo group[43,44]
Dextrose prolotherapy	Symptomatic improvement in pain, functionality, and stiffness	Pain at injection site, risk of bleeding, and infection appear similar to corticosteroid injections	Statistical improvement in pain, function, and stiffness compared with saline injection at 26 and 52 weeks; more data needed to assess efficacy[45,46]
Platelet-rich plasma (PRP) injections	Augmentation of tissue healing, symptomatic improvement in pain and functionality	Pain at injection site, risk of bleeding, and infection appear similar to corticosteroid injections	Newer modality with limited clinical evidence; 2 RCTs showed better clinical outcomes 24 weeks after injection compared with hyaluronic acid (HA); meta-analysis of 16 studies showed PRP more effective than HA at 12 months[47,48]

GAIT, Glucosamine/chondroitin Arthritis Intervention Trial; GI, gastrointestinal; NIH, National Institutes of Health; NSAIDs, nonsteroidal anti-inflammatory drugs; OA, osteoarthritis; RCT, randomized controlled trial.

for patients who have severe pain and are not candidates for surgery. Initial opioid therapy, for no more than 2 weeks, may include low-potency agents such as codeine, 30 mg every 6 hours, or tramadol, 50 mg 3 times daily, as needed. Rarely, more potent opioids may be required, but these should be used short term for exacerbations.[28]

Adjunctive topical therapy. As an adjunct to oral analgesia, diclofenac 1% gel has shown efficacy when applied 4 times daily at 4 g/application.[8] Similarly, capsaicin 0.0025% cream applied to the knee 4 times daily can improve subjective reports of pain.[37]

The ACR recommends starting patients ages ≥75 years on a topical rather than oral NSAID.[33] Diclofenac 1% gel is recommended first, with capsaicin cream to be added if needed.[8,10]

Other therapies. Intra-articular injection of sodium hyaluronate has been shown to be effective for symptomatic relief of knee OA by increasing viscoelasticity of synovial fluid and decreasing degeneration of articular cartilage. Injections typically are given once weekly for 3 to 5 weeks.[38] Colchicine, glucosamine/chondroitin supplements, S-adenosylmethionine (SAMe) supplements, and other agents have been studied in the treatment of OA. Evidence of their safety and effectiveness is shown in TABLE 2.[39-48]

Glucocorticoid injections may provide relief for 4 to 6 weeks

If patients continue to have significant pain or are not candidates for NSAIDs or other analgesic therapy, glucocorticoid injections may be considered. Injections typically are tried prior to referral for surgery.

Early studies showed glucocorticoid injections for OA knee pain generally resulted in clinical relief for 4 to 6 weeks.[49] Newer data

suggest that ultrasound guidance of joint injections—including those for the knee—improves accuracy, leads to great improvement in joint function, reduces procedure pain, and improves pain scores 4 to 6 weeks after injection.[50,51] Triamcinolone hexacetonide 20 mg or methylprednisolone acetate 40 mg are typically used (TABLE 1); mixing the glucocorticoid with lidocaine 1% or bupivacaine 0.025% can provide immediate relief after injection.[38]

Expert opinion once suggested giving glucocorticoid injections for OA no more frequently than 3 times per year in the same joint because of concerns about injury to intra-articular structures.[28] Recent data, however, suggest long-term safety of more frequent injections. In a randomized controlled trial, 68 patients with knee OA who received 4 injections of triamcinolone acetonide per year for 2 years showed no deleterious effect on cartilage depth in the injected knees. Patients receiving the corticosteroid injections showed significant improvements in knee pain and stiffness as compared with controls injected with saline.[52]

Time to consider joint osteotomy or total knee arthroplasty?

If conservative therapies fail to ameliorate pain, the patient may benefit from referral for surgical evaluation. Surgical options for OA include knee arthroscopy, joint osteotomy, and total knee arthroplasty (TKA). Most patients who undergo TKA report improved overall function and decreased pain, although the success rate depends on severity of disease, the surgeon's experience, and postoperative rehabilitation.[28]

CASE

Mr. D experiences significant relief from today's ultrasound-guided glucocorticoid injections to his thumb and knee, but he understands that the OA symptoms will recur. He talks with you about his continuing pain and functional limitations, despite having tried conservative treatments for 4 years. He now recognizes that he may benefit from surgery and is committed to referral to discuss the surgical options for his thumb and knee.

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