

# Osteoarthritis

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## Pathophysiology

Osteoarthritis (OA) is the most common form of arthritis. There is no universally accepted definition of OA, but it is characterized by abnormalities in the synthesis and degradation of articular cartilage in synovial joints. It is estimated to affect 1 in 10 Canadian adults.<sup>1</sup> In the elderly, it ranks second to cardiovascular disease in terms of causing chronic disability.<sup>2</sup>

OA results from damage and destabilization of synovial joints. Synovial joints are structures in which the opposing bony surfaces are covered with a layer of cartilage. There is also a joint cavity containing synovial fluid and lined with synovial membrane.<sup>3</sup> Cartilage acts as a shock absorber and, with synovial fluid, provides a smooth, low-friction surface for movement. Surrounding the joints is the articular capsule, ligaments, muscles and tendons, all of which act to stabilize and protect the joint.

Synovial joints are present in fingers, wrists, ankles, knees and hips. In a normal joint, cartilage is in a continuous process of formation and degradation. In OA, two processes occur which lead to joint destruction. First, there is progressive cartilage breakdown without adequate formation, leading to thinning of the articular cartilage and instability of the joint. Second, *osteophytes* or bony outgrowths appear. These represent new bone formation in areas away from the area of cartilage destruction. Although the role of osteophytes is unclear, they may act in part as stabilizers in response to joint destruction.

The most significant risk factor for OA is advancing age. Other risk factors include obesity, quadriceps muscle weakness, genetic susceptibility, major trauma and joint overuse or injury associated with certain sports and occupations.<sup>4</sup>

It is estimated that by age 65, up to 70% of people will have radiographic evidence of OA. However, only one-third of them will complain of symptoms.

## Goals of Therapy

- Relieve symptoms
- Maintain or improve mobility
- Minimize functional disability and improve physical functioning
- Educate patients and caregivers to assist them to understand their condition and make informed decisions about which therapies to choose

## Patient Assessment

Joint pain is the most common symptom of OA. The pain does not arise from the damaged cartilage itself, but is the result of the various stresses placed on the muscles, ligaments and tendons in the areas surrounding the cartilage as a result of the damage. Stiffness after inactivity and limited range of motion are other common symptoms. Inflammation may or may not be present. Crepitus may be present with joint movement. Table 1 lists the signs and symptoms of OA.

Rheumatoid arthritis is a systemic inflammatory disease that often presents with joint pain as one of many symptoms. The scope of this chapter does not include the management of rheumatoid arthritis; however, symptom recognition is important so that patients with suspected rheumatoid arthritis can be referred to a physician for a full work-up and appropriate therapy to control inflammation and delay disease progression (Table 1).

Since joint pain can have a number of causes, it is important that more serious conditions requiring

medical intervention be ruled out. In particular, recent history of significant trauma, hot, swollen joints and signs and symptoms of infection should prompt a physician referral.

Reports of arthralgia have occurred with numerous drugs; however, the numbers are small and often a cause and effect relationship cannot be clearly established. There are also a number of medications that have been implicated in drug-induced systemic lupus erythematosus (SLE), of which arthralgia is often a presenting symptom. Medications associated with SLE include: acebutolol, carbamazepine, chlorpromazine, hydralazine, isoniazid, methyldopa, minocycline, penicillamine, procainamide, quinidine and sulfasalazine.<sup>6</sup> Fortunately, these arthralgias usually resolve with discontinuation of the offending agent.

Figure 1 shows an algorithm for assessing patients with joint pain.

## General Principles of Therapy

Current pharmacologic therapies for OA provide symptomatic relief but are not curative. Therefore, the

choice of agent is based on a combination of risk versus benefit, cost and patient preference. Pharmacologic therapy should always be initiated in combination with nonpharmacologic modalities. If tolerated, pharmacologic therapies (except localized therapy) should be tried for at least two to four weeks in order to allow the patient to fully assess its effectiveness.

## Nonpharmacologic Therapy

Nonpharmacologic therapy (Table 2) should always be initiated first or started concurrently with drug therapy. The quality of published evidence supporting these modalities is varied. There is reasonably good evidence to support the use of exercise,<sup>8</sup> exercise plus physiotherapy,<sup>22</sup> TENS<sup>19</sup> and patient education for self-management and social support.<sup>11-14</sup>

Surgery is usually reserved as a last resort for patients with severe, painful and activity-limiting OA who have tried other pharmacologic and nonpharmacologic modalities.<sup>17</sup>

Table 1: **Clinical Features of Osteoarthritis versus Rheumatoid Arthritis**<sup>5</sup>

Feature	Osteoarthritis	Rheumatoid arthritis
<b>Symptoms:</b>		
Stiffness	Morning or after periods of inactivity; usually lasts < 30 min	Significant, prolonged (> 60 min) in the morning
Symptoms localized	Yes – limited to affected joints	No
Pain	Worsens with activity or after prolonged use (especially with weight bearing activity).	Worsens after prolonged inactivity; usually improves with activity.
<b>Signs:</b>		
Symmetry	Occasional	Common
Tenderness	Unusual	Over entire exposed joint spaces.
Inflammation	Unusual	Common
Instability	Occasional; buckling or joint instability can result in decreased range of movement and falls.	Uncommon
Multisystem disease	No	Often feel systemically unwell (e.g., can have one or more of fatigue, fever, chills, weight loss, hair loss, dry mouth or dry eyes)

Figure 1: Assessment of Patients with Joint Pain<sup>3,7</sup>

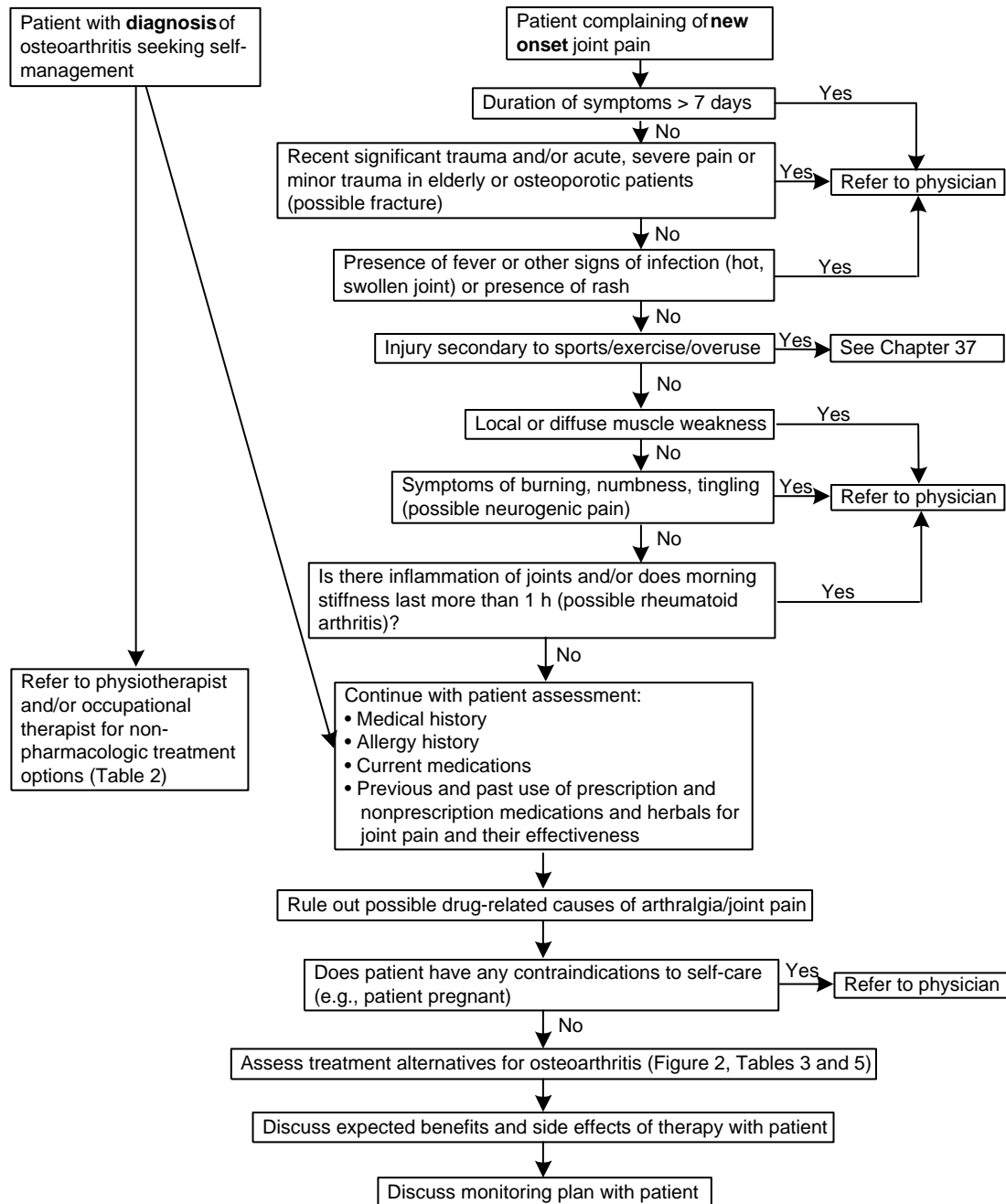


Table 2: **Nonpharmacologic Therapy for Osteoarthritis**

Modalities supported by evidence	Purpose/Benefits
Strength training and aerobic exercise <sup>8,9,10</sup>	<ul style="list-style-type: none"> <li>• Reduce pain</li> <li>• Preserve flexibility</li> <li>• Improve range of motion and strength</li> <li>• Decrease need for analgesic medication and decrease physician visits</li> </ul>
Patient education for self-management <sup>11</sup>	<ul style="list-style-type: none"> <li>• Reduce pain</li> <li>• Decrease frequency of physician visits</li> <li>• Overall improvement in quality of life</li> </ul>
Social support (e.g., telephone follow-up); <sup>12,13</sup> education of family members and caregivers <sup>14</sup>	<ul style="list-style-type: none"> <li>• Improve pain and functional status</li> </ul>
Use of ambulation aids and assistive devices (e.g., canes, grab bar, raised toilet seat) <sup>15,16</sup>	<ul style="list-style-type: none"> <li>• Improve functional status and ambulation</li> <li>• Facilitate carrying out activities of daily living</li> </ul>
Joint protection (e.g., splints, braces) and energy conservation (e.g., avoidance of stair climbing, take short rest periods, pace activities) <sup>17</sup>	<ul style="list-style-type: none"> <li>• Minimize pain</li> <li>• Improve joint function</li> </ul>
Weight loss in obesity (i.e., reduction in body fat) <sup>18</sup>	<ul style="list-style-type: none"> <li>• Decrease load on weight bearing joints</li> <li>• Improvement in symptoms in OA of knee</li> </ul>
Corrective footwear <sup>17</sup>	<ul style="list-style-type: none"> <li>• To correct abnormal biomechanics secondary to OA knee</li> </ul>
Surgery <sup>17</sup>	<ul style="list-style-type: none"> <li>• Indicated in patients with severe symptomatic OA who have failed nonsurgical strategies and continue to have significant limitations in activities of daily living</li> </ul>
TENS (transcutaneous electrical nerve stimulation) <sup>19</sup>	<ul style="list-style-type: none"> <li>• Pain control in OA knee</li> </ul>
Acupuncture <sup>20</sup>	<ul style="list-style-type: none"> <li>• May improve pain, not function in OA knee</li> </ul>
<b>Lack of evidence to support use of these modalities</b>	
Heat, cold, massage	<ul style="list-style-type: none"> <li>• Lack of well-designed randomized controlled trials</li> <li>• Anecdotal evidence suggests alleviation of pain</li> <li>• Massage produces counterirritation</li> </ul>
Low level laser <sup>21</sup>	<ul style="list-style-type: none"> <li>• Lack of evidence and inconsistencies between trials to recommend use in OA</li> </ul>

## Pharmacologic Therapy

### Nonprescription Agents (Table 3)

#### **Acetaminophen**

The American College of Rheumatology recommends acetaminophen as the initial drug of choice for symptomatic relief of OA.<sup>17</sup> The rationale for using acetaminophen is largely based on the fact that it is relatively safe, well tolerated and easily accessible. Acetaminophen and NSAIDs are comparable in their

ability to provide pain relief in mild to moderate OA of the knee.<sup>26,27</sup> There are no published trials of acetaminophen in OA of the hip.

Maximum therapeutic doses should be tried for an adequate period (two to three weeks) in order to assess efficacy. Conditions such as chronic alcohol abuse and liver disease preclude the long-term use of maximum therapeutic doses and should be investigated prior to beginning therapy.

Table 3: Nonprescription Therapy of Osteoarthritis<sup>3,7,23,25,26</sup>

Drug	Therapeutic effects and usual dose	Onset of action	Adverse effects/Precautions	Clinically significant drug interactions	Comments
<b>Oral analgesics:</b>					
Acetaminophen	325-1000 mg q4-6h SR: 650 mg q8h (max 4 g per day)	Maximal onset of pain relief within 24-48 h	Hepatotoxicity – increased risk in patients with excessive alcohol intake (> 3 drinks per day), malnourishment or pre-existing hepatic disease. Baseline LFTs should be measured in high-risk patients.	Alcohol (see precautions). Warfarin (with regular use of > 2 g per day acetaminophen). Phenytoin (may increase metabolism of acetaminophen and formation of toxic metabolite thereby increasing the risk of hepatotoxicity; risk may be higher in patients taking high therapeutic doses of acetaminophen and phenytoin chronically. Interaction has not been well-documented).	Lower doses may be required in patients with severe hepatic and renal disease. Caution with concurrent use of acetaminophen-containing OTC products (do not exceed 4 g per day). Continuous therapy should be considered in individuals with pain persisting throughout the day. PRN dosing is acceptable for episodic pain of short duration.
NSAIDs: ASA Ibuprofen	Ibuprofen: 200-400 mg q6-8h; usual maximum daily dose: 3200 mg per day. ASA: 325-650 mg q6-8h; usual maximum daily dose: 3900 mg per day (not recommended for use if CrCl < 10 mL/min)		Local GI effects: dyspepsia, diarrhea. GI complications: ulceration/upper GI bleed. CHF may be exacerbated by use of NSAIDs. Renal effects: more likely in elderly or patients with pre-existing renal disease or comorbid conditions that may affect renal function (e.g., diabetes, CHF, hypertension). ↑ LFTs: transient; hepatotoxicity is rare; more likely to occur in patients with pre-existing hepatic disease or in patients with excessive alcohol intake (> 3 drinks per day).	Warfarin (↑ bleeding risk); monitor INR more frequently during initial period after NSAID started and watch for signs of bleeding. ↑ Lithium levels – monitor levels. ↑ Methotrexate levels (rare) – monitor for toxicity. Antihypertensives (e.g., beta-blockers, diuretics, ACEI): possible ↓ in antihypertensive effects. Measure baseline BP, then remeasure in 1-2 wk and adjust antihypertensive therapy as required.	NSAIDs are not a substitute for ASA being taken for MI or stroke prophylaxis. Avoid concurrent use of NSAID-containing OTC products (increased risk of GI-related side effects). Continuous therapy should be considered in individuals with pain persisting throughout the day. PRN dosing is acceptable for episodic pain of short duration. Consider prophylaxis with misoprostol or proton pump inhibitor in high risk patients (Table 4). Avoid NSAIDs in patients with ASA or ibuprofen hypersensitivity.

Acetaminophen + caffeine + codeine 8 mg	1-2 tablets q4-6h (maximum 4 g acetaminophen per day)		See acetaminophen. Sedation, nausea, vomiting, constipation	See acetaminophen. Concurrent use of other sedating or constipating medications	Recommended for short-term use only only (e.g., 2-3 days). Elderly are at increased risk for adverse effects.
<b>Alternative therapies:</b>					
Glucosamine sulfate Glucosamine HCl Chondroitin sulfate	Glucosamine sulfate 500 mg TID (more extensively studied than HCl product). Chondroitin 400 mg TID	Onset: 1-3 wks	Nausea, dyspepsia, diarrhea. Glucosamine products derived from marine exoskeletons might cause allergic reactions in people with shellfish allergies. To date, no such reactions have been documented.		Long term efficacy and toxicity are unknown. Lack of product standardization may result in inter- and intra- product variation. Less evidence for efficacy of chondroitin compared to glucosamine. Awaiting results of study sponsored by National Institute of Health (NIH) comparing monotherapy of each and combination therapy.

SR = *sustained release*

**NSAIDs**

Only ibuprofen and ASA are available without a prescription in Canada. Both the American College of Rheumatology<sup>17</sup> and the American Geriatrics Society<sup>28</sup> recommend NSAIDs as second-line therapy after failure of acetaminophen in management of OA pain. Although acetaminophen is superior in terms of safety, NSAIDs are preferred by OA patients due to better pain relief.<sup>29,30</sup> Not surprisingly, patients who discontinue NSAID use due to toxicity are less willing to resume therapy with another NSAID.<sup>29</sup>

In terms of which NSAID is the most efficacious or safest in OA, there are no definitive data to indicate the superiority of one over another. Systematic reviews have consistently identified lack of standardization in case definition of OA, outcome assessments<sup>31</sup> and failure to use therapeutically equivalent doses<sup>32</sup> as limitations to being able to draw any conclusions.

GI complications are the side effects of greatest concern with NSAID therapy.

Common local GI effects such as nausea and dyspepsia occur in approximately 10 to 20% of NSAID users and are relatively minor in clinical significance. More serious GI complications such as perforated ulcers and hemorrhage have been estimated to occur in OA patients at an incidence of less than 1% per year.<sup>33</sup> Although the incidence appears low, because NSAIDs are one of the most commonly prescribed drug classes, a relatively large number of patients is affected. Unfortunately, the presence of local GI symptoms such as dyspepsia is not a predictor for serious GI complications and vice versa. There is also no definitive evidence to show that one NSAID has an advantage over another in terms of causing GI complications. However, there are several factors that increase a patient's risk for the development of upper GI adverse events (Table 4).

Prior to starting NSAID therapy, patients at risk of developing serious NSAID-related GI complications should be identified and preventive measures taken. H<sub>2</sub>RAs and antacids provide relief from dyspeptic symptoms, but not against more serious GI complications. Misoprostol and proton pump inhibitors such as omeprazole are both appropriate options for preventing serious GI complications such as gastric ulcer, perforation, gastric outlet obstruction or bleeding.<sup>25,34,35</sup>

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**Table 4: Risk Factors for the Development of Upper GI Adverse Events<sup>17,33</sup>**

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**Established:**

Age ≥ 65 yrs  
History of ulcer  
Concomitant use of corticosteroids, anticoagulants  
Use of higher doses of NSAIDs  
Use of more than one NSAID  
Presence of multiple systemic disorders

**Possible:**

Concomitant infection with *Helicobacter pylori*  
Cigarette smoking  
Regular alcohol consumption

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**Combination Products**

Numerous combination analgesic products containing codeine 8 mg are available OTC. The effectiveness of using low-dose combination codeine products compared with single ingredient products (e.g., NSAIDs, acetaminophen) for OA has not been adequately studied.

**Topical Counterirritants** (Table 5)

Although there is little evidence to support their use in OA, topical agents are a reasonable alternative for patients who cannot tolerate or are reluctant to use systemic agents. They may also be tried as an adjunct to systemic agents where pain relief is not adequate.

**Methyl salicylate** and **trolamine salicylate** act as counterirritants and analgesics. Methyl salicylate has not been shown to improve symptoms in osteoarthritis. One study found trolamine salicylate was significantly better than placebo in improving pain and stiffness in patients with OA of the hands.<sup>36</sup> Since they are readily available OTC, the potential exists for overuse of these products leading to salicylate toxicity.<sup>37</sup>

The evidence for using **capsaicin** in OA is limited to a few small, short-term studies<sup>38-40</sup> and one meta-analysis.<sup>41</sup> Compared with placebo, capsaicin has been shown to decrease pain symptoms over a 4 to 12 week period in patients with OA of the hand<sup>40</sup> or knee.<sup>38,39</sup> In these studies, concomitant use of acetaminophen<sup>38</sup> or NSAIDs<sup>39,40</sup> was permitted. Capsaicin's role in OA management may be more as adjuvant therapy after unsatisfactory trials of acetaminophen or NSAIDs. Unfortunately, the tingling and

burning sensation caused by capsaicin often prevents an adequate trial of this medication.

### Alternative Agents

**Glucosamine** and **chondroitin** are endogenous substances that are responsible for maintaining the integrity of cartilage within a joint. Exogenous formulations (i.e., glucosamine sulfate, glucosamine hydrochloride, chondroitin sulfate) have been evaluated in the treatment of OA primarily for their pharmacologic effect that mimics their physiologic effect on cartilage tissue. The proposed mechanisms of action are to stimulate the production of cartilage, prevent cartilage destruction by inhibiting inflammatory mediators and/or enzymes, and maintain viscosity of the joint.<sup>42,43</sup> This may result in positive outcomes such as a decrease in pain and an increase in function. In addition, preliminary studies suggest these agents may

possess chondroprotective effects resulting in a decrease in joint space narrowing.<sup>44</sup> However, this finding needs to be further validated and quantified in larger studies.<sup>45</sup>

Evidence suggests that these agents *may* be beneficial in decreasing pain and increasing mobility in the short-term.<sup>46,47</sup> However, limitations with respect to study design, publication bias, long-term efficacy and safety, and product variation may influence the individual results seen with these agents.<sup>17</sup> As a result, these agents should not be recommended as first-line therapy.

### Prescription Medications

#### COX-2 Inhibitors

**Celecoxib** and **rofecoxib** are as effective as NSAIDs for pain control in OA of the hip and knee<sup>48</sup> and are

Table 5: **Nonprescription Topical Counterirritants for Osteoarthritis**<sup>3,23</sup>

Drug	Dose and onset	Adverse effects/Precautions	Drug interactions (clinically significant)	Comments
Methyl salicylate Trolamine salicylate	For all products: Apply up to 3-4 times per day. Onset of maximal effect: 2-4 wks (with continuous usage)	Avoid in ASA allergic patients. Use with caution in patients with conditions associated with ASA sensitivity (i.e., severe asthma or nasal polyps). Salicylate toxicity may occur if applied over large surface area or on broken skin.	Warfarin: Potentiation of anticoagulant effects; monitor INR more frequently and watch for signs of bleeding if ongoing concurrent use (especially if topical product is being applied over a large area).	For all products: Do not apply near mucous membranes or on broken skin. Do not cover with tight or occlusive dressing. Do not place heating devices (e.g., hot water bottle, heating pad) on skin after application of product.
Menthol		Discontinue if hypersensitivity occurs (signs include irritation, rash, burning, stinging, swelling or infection).		
Capsaicin	Capsaicin should be applied 3-4 times per day continuously for 2-4 wks to achieve maximum therapeutic effect	Apply with gloves and wash hands thoroughly after application to avoid irritation of other areas. Tingling, burning or redness will occur in the majority of patients. Usually decreases within 72 h with repeated use; if effect is bothersome, pre-treatment with topical lidocaine or EMLA <sup>®</sup> cream may help.		

associated with a lower incidence of gastroduodenal ulcers compared to NSAIDs.<sup>49,50</sup> The comparative safety of COX-2 inhibitors versus NSAIDs plus cytoprotection has not been evaluated. Similar to NSAIDs, COX-2 inhibitors can exacerbate pre-existing renal disease. Baseline and periodic monitoring of creatinine clearance and electrolytes should be carried out in high-risk patients.

### Localized Therapy

The use of **intra-articular corticosteroids** in OA is limited to acute knee pain and patients who have local signs of inflammation and joint effusion. If joints are painful and swollen, aspiration of fluid followed by intra-articular injection of a glucocorticoid (e.g., triamcinolone hexacetonide) is effective in temporarily (i.e., four to six weeks) decreasing pain and increasing quadriceps strength.<sup>7</sup> It is often used in combination with other therapies, although it can be used as monotherapy. Repeated injections may damage cartilage. As such, it is recommended that the same site not be injected more than three to four times per year.<sup>7,17</sup>

The efficacy of intra-articular corticosteroids for hip OA has not been studied and use is not routinely

recommended because of the risk of cartilage damage through repeated injections.<sup>15</sup>

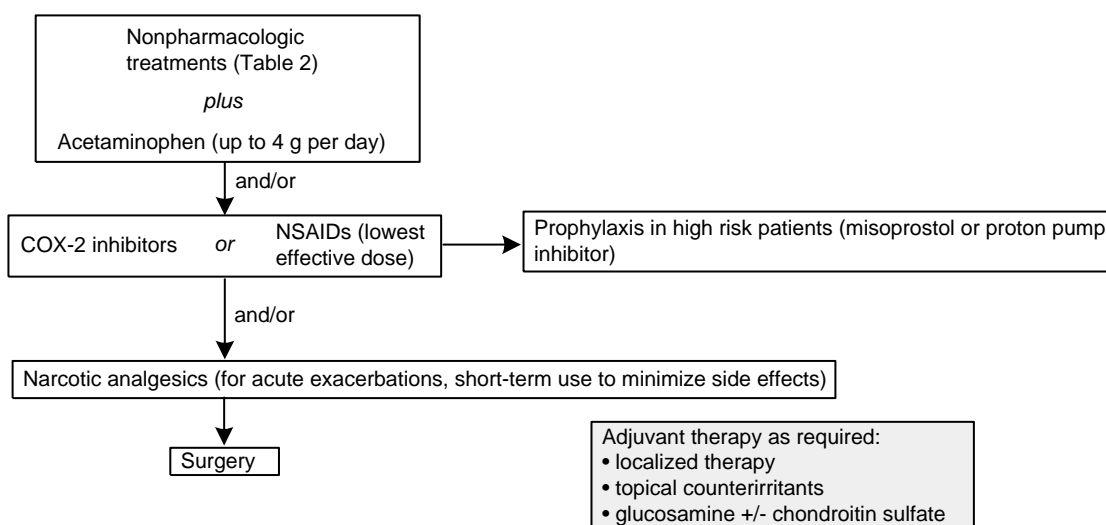
**Hyaluronan** is a linear polysaccharide found in synovial fluid. Three to five weekly injections have modest pain relieving effects in patients with mild to moderate OA of the knee.<sup>7</sup> The onset of pain relief is slower than with corticosteroids, although the effect may last longer. The effect of repeated courses of hyaluronan injections is unknown. The injections are expensive, costing over \$200 for three injections. The use of these products is usually reserved for those patients who have failed other therapies.

### Narcotic Analgesics

In patients who do not respond to other analgesics or experience acute exacerbations of OA pain, a narcotic analgesic used alone or in combination with acetaminophen or NSAIDs may be useful.<sup>17</sup> However, side effects such as sedation, constipation, tolerance and dependence limit the long-term use of these agents.

Figure 2 summarizes various osteoarthritis treatment options.

Figure 2: Algorithm for Treatment of Osteoarthritis



## Monitoring of Therapy

Table 6 provides a monitoring plan for patients with osteoarthritis.

## Advice for the Patient

Counsel patients on:

- The importance of using nondrug measures concurrently with medication therapy;
- Expected benefits of therapy;

- Possible side effects and their management;
- When to contact a physician.

## Resource Tips

For more information on OA contact:  
 The Arthritis Society (National Office)  
 393 University Avenue, Suite 1700  
 Toronto, Ontario M5G 1E6  
 Canada  
 Tel.: 1-800-321-1433  
 Web site: www.arthritis.ca

Table 6: **Monitoring Therapy of Osteoarthritis**

Parameter	Degree	Timeframe	Action/Comments
Pain relief	Elimination or improvement toward predefined goals as set by patient	<b>Patient:</b> assess daily <b>Pharmacist:</b> phone call on days 3, 7, 14 and 28	Refer to physician if not receiving symptom relief after adequate trial of at least two analgesics or on development of new or worsening pain during therapy. Considerations: • Use visual analog scale or other individual measure to quantify and characterize pain (e.g., ADLs – walking, gardening) • Establish acceptable level of pain control and functioning with the patient at the beginning of therapy • Considerations in timing of medications: –Around-the-clock vs. PRN –Take dose of analgesic at least 1 h prior to activities that may exacerbate pain.
Nausea, dyspepsia, abdominal discomfort	Minimal or none during therapy	<b>Patient:</b> monitor daily <b>Pharmacist:</b> phone call on days 3 and 7	Change therapy if symptoms severe or intolerable. Minimize development by advising to take with food or milk. Assess risk of GI complications (Table 4). If high risk, refer to physician. Otherwise, consider antacids or OTC H <sub>2</sub> RAs to treat dyspepsia.
Hematemesis, melena, hematochezia	None during therapy	<b>Patient:</b> monitor daily on an ongoing basis <b>Pharmacist:</b> phone call on days 3 and 7	Patient should seek medical attention if these signs or symptoms develop. Discontinue therapy immediately.
Renal function and signs of fluid retention (e.g., weight gain or edema) in high risk patients (ASA or ibuprofen—see Table 3)	No significant change in renal function	<b>Physician:</b> Serum creatinine and Na <sup>+</sup> , K <sup>+</sup> at baseline, q1-2 week × 3 then periodically afterwards. Patient to monitor for signs of fluid retention on an ongoing basis (e.g., edema; patients with severe CHF should measure their weight daily).	Discontinue NSAID if significant change in serum creatinine or electrolytes or signs of fluid retention.

ADL=activities of daily living; H<sub>2</sub>RA = H<sub>2</sub>-receptor antagonist

**Suggested Readings**

- American College of Rheumatology Subcommittee on Osteoarthritis Guidelines. Recommendations for the medical management of osteoarthritis of the hip and knee: 2000 update. *Arthritis Rheum* 2000;43(9):1905-15.
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## Osteoarthritis—Patient Information

### **What is osteoarthritis?**

Osteoarthritis (OA) is also known as degenerative joint disease. It is the most common kind of arthritis. It usually affects the joints of the neck, lower back, knees, hips and fingers of middle-aged and older people.

### **What causes osteoarthritis?**

OA results from breakdown of joint cartilage. This can happen for many reasons. Some kinds of OA are passed from one generation to the next. In most people, OA occurs when the cartilage that normally cushions and protects the joints becomes worn down or works less efficiently. The wear and tear on the joints may also occur in joints that have suffered previous injury or been subjected to prolonged heavy use.

### **What are the signs and symptoms of osteoarthritis?**

People with OA experience pain, stiffness and poor function in or around the joint. Sometimes a grating sound can be heard when moving the joint.

OA can be diagnosed by your doctor using a combination of X-rays, physical examination and ruling out other types of arthritis.

### **The goals of treatment are to:**

- Decrease pain and discomfort so that you can continue to carry out your usual daily activities;
- Protect your joints.

### **Always try non-drug measures first and combine them with drugs if needed.**

### **Non-drug measures:**

- Consult a physiotherapist and/or occupational therapist for tips on exercises and devices to reduce pain, strengthen and protect the affected joints.
- Consider weight loss if you are overweight to relieve stress on your joints.
- Learn as much as you can about OA to improve your knowledge about how best to manage pain and protect your joints.

### **Nonprescription medications:**

There are several types of medications available for helping with OA pain. If you need to use pain medication for more than seven days in a row, talk to your doctor to see if this is the best treatment for you.