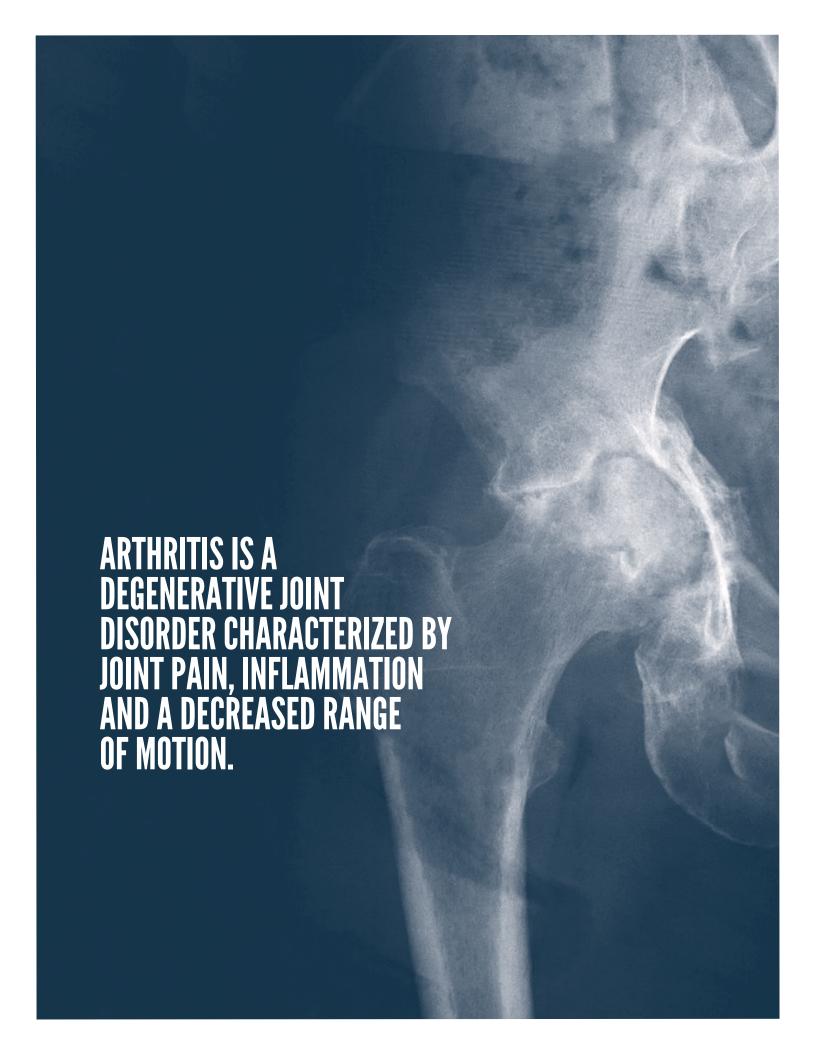
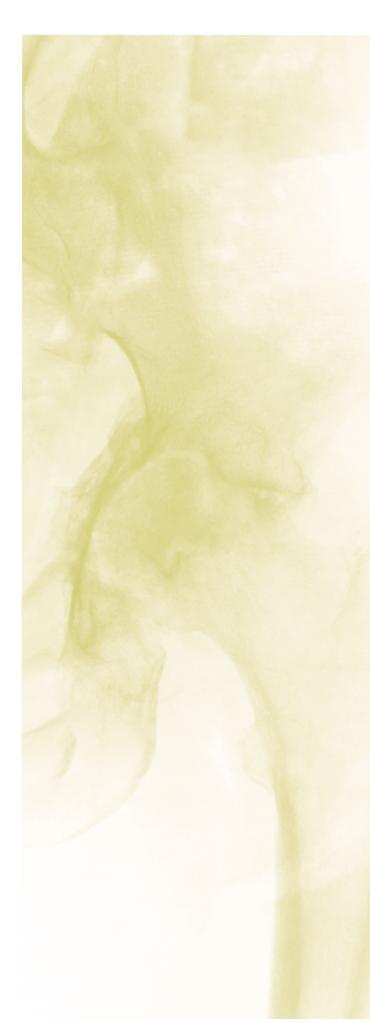
WHAT YOU NEED TO KNOW MANAGING ARTHRITIS IN OLDER PERSONS







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rthritis is a joint disorder characterized by inflammation and often results in joint pain referred to as arthralgia. There are over 100 types of arthritis characterized to date, and together they are among the most common illnesses.

The most common type of arthritis, **Osteoarthritis** (**OA**), affects an estimated 1 in 10 Canadians and is also the most common musculoskeletal disorder worldwide. OA is a progressive disease of the synovial joints that causes breakdown of cartilage and bone, which ultimately results in pain, stiffness and functional disability. OA was previously believed to be a degeneration of the joints due to daily "wear and tear". While the previous theory holds some truth, it is now defined as a systemic biochemical and inflammatory disorder resulting from an imbalance of joint destruction and repair.

Rheumatoid Arthritis (RA) is a systemic autoimmune disease causing chronic inflammation of the joints. RA is a symmetric and erosive polyarthritis that results in pain, stiffness and deformity of the joints. While the disease is characterized by involvement of the joints, it is also associated with inflammation and injury of most internal organs. Patients with RA have an increased risk of cardiovascular disease, lymphoproliferative disease, depression and premature mortality.

WHAT ARE THE RISK FACTORS?

OA can impact any joint but most commonly manifests in the spine, hands, hips or knees. Primary OA is idiopathic and most frequently affects the elderly population. Secondary OA is the result of some type of insult to the joint (eg. trauma, infection) or other conditions such as gout or rheumatoid arthritis. The most at-risk population includes the elderly and female gender. Other risk factors may include obesity, weak quadriceps muscles, family history, and joint overuse or injury.

RA onset can occur at any age, but most frequently at age 40-50. RA affects women three times more than men. The cause of this rheumatic disease is unknown, but it is suspected that it may have a hereditary or environmental component.

WHAT ARE THE SIGNS AND SYMPTOMS?

The damage to cartilage that characterizes **OA** most commonly results in asymmetrical joint pain. The damaged cartilage in the synovial joints manifests pain through stress placed on muscles, ligaments and tendons in the surrounding areas. Pain, stiffness and limited range of motion at the joint are the most common symptoms.

Patients with **RA**, on the other hand, exhibit swelling in symmetric joints of the hands and feet. However, early in the disease, the symptoms and diagnostic images may not be typical of the disease. Like other autoimmune disorders, there are periods where the disease is active causing flares and symptoms, and periods of inactivity (remission).

HOW IS IT DIAGNOSED?

OA is most commonly identified through patient history and can be confirmed by diagnostic imaging. Joint stiffness symptomatic of OA often lasts less than 30 minutes and usually presents in the morning. X-ray and diagnostic ultrasound can confirm the presence and extent of joint damage, however, the extent of damage displayed in imaging often does not directly translate to clinical symptoms.

The diagnosis of **RA** is based on history, physical examination, serology or radiographic imaging.

HOW IS IT TREATED?

The focus of treatment in both **OA** and **RA** is to improve quality of life and decrease symptoms.

In **OA** the primary focus of treatment is to decrease pain, improve joint function and prevent further damage. Non-pharmacological lifestyle changes should be the primary treatment, or they can be used to compliment initial medication therapy. Aerobic exercise, strength training, physiotherapy, weight loss and orthotic support devices can improve mobility and decrease pain. Surgical intervention is a last resort in severe OA where pharmacological and non-pharmacological therapies have failed

There are a variety of pharmacological therapies available without a prescription for initial OA treatment. A step-wise approach to therapy including topical analgesics, oral analgesics and lastly injectable analgesics is recommended. Topical non-steroidal anti-inflammatory drugs (NSAIDs), such as diclofenac, are recommended prior to oral medications, especially in the elderly who are at high risk of NSAID side effects. Capsaicin has shown some benefit, but the burning sensation and risk of transferring the medication to mucous membranes and other areas often make it an unsuitable choice.

Oral acetaminophen is the first oral drug of choice due to the favourable side effect profile. The maximum recommended dose

is 4g/day, however the American Geriatric Society recommends 2-3g/day with hepatic insufficiency. NSAIDs are often more effective in pain reduction, however they carry an increased risk of a number of side effects including serious GI complications (such as ulcers and hemorrhage), hypertension, edema, congestive heart failure and impaired renal function. The use of opioids is generally recommended for severe disease or in combination with other medications due to potential increased risk of constipation, drowsiness and dizziness. This is an unfavourable side effect profile, especially in the elderly which can greatly increase risk of falls and injury. Although we know that glucosamine and chondroitin maintain the integrity of cartilage within the joint, exogenous administration does not translate to pain reduction in OA. Intra-articular corticosteroid and hyaluronic acid injections are effective in reducing pain and stiffness. The corticosteroid injections are most effective after 1 to 2 weeks and may last up to 3 to 6 months. The hyaluronic acid injections may have a longer duration.

RA treatment ideally encompasses a multidisciplinary approach that involves the patient, family and health care professionals. Rest, appropriate activity and exercise and methods of pain modulation are pivotal to the success of treatment. Drug therapy includes conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs or traditional/non-biologic agents), biological DMARDS (bDMARDs) and targeted synthetic DMARDs (tsDMARDs). After diagnosis is made and confirmed with a rheumatologist, a csDMARD, ideally methotrexate (MTX) should be initiated.

Other options in patients with a MTX contraindication include sulfasalazine, leflunomide or hydroxychloroquine. Combination therapy including MTX is a second treatment option. The bDMARDs target key mediators of inflammatory synovitis and bone and cartilage destruction. Tumour necrosis factor inhibitors (TNFi) include infliximab, etanercept, adalimumab, certolizumab and golimumab. Interleukin (IL) inhibitors including tocilizumab and anakinra have a different mechanism of action and can be used in combination with other therapies or as monotherapy after failure to csDMARDs and/or TNFi therapy. Other biologics including abatacept and rituximab are reserved for patients with inadequate response to one or more csDMARDs and/or TNFi therapy.

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SOURCES

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