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Osteoarthritis Medication and Overview: A Narrative Review

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ABSTRACT

Osteoarthritis, the most common form of joint disease, is primarily a disease of aging. Ninety percent of all people have radiographic features of osteoarthritis in weight-bearing joints by age 40. Symptomatic disease increases with age. Risk factors for this disorder include gender, genetics, obesity, and work history. Osteoarthritis develops in women more often than in men. This arthropathy is characterized by cartilage degeneration and bony hypertrophy at the articular margins. Inflammation is usually minimal. Hereditary and mechanical factors may be involved in the pathogenesis. This review provides an overview of counseling osteoarthritis and its treatment and management.

Introduction

Degenerative joint disease is divided into two types, primary and secondary.[1] Primary degenerative joint disease generally affects some or all of the following; the proximal interphalangeal (PIP) joints of the fingers, the carpometacarpal joints of the thumb, hip, knee, metatarsophalangeal (MTP) joint of the big toe, and the cervical and lumbar spine.[2] Meanwhile, the secondary degenerative joint disease can occur in any joint as a sequel to articular injury. Injuries may be acute, as in fractures; or chronic, due to overuse of joint or metabolic diseases, for example in hyperparathyroidism, hemochromatosis, ochronosis, or joint inflammation (rheumatoid arthritis).

Osteoarthritis has a dangerous onset. Initial symptoms may include articular stiffness, rarely lasting more than 15 minutes. This symptom progresses to pain on movement of the affected joint, worsened by activity or weight-bearing and relieved by rest.[3] Flexion contractures or varus knee deformities are not uncommon, and the enlarged bones of the DIP (Heberden node) and PIP (Bouchard node) are occasionally prominent (figure 1). There is no ankylosis, but the limitation of movement of the joint or the affected joint is common. Crepitus may often be felt over the knee. Joint effusion and other signs of articular inflammation are mild. However, in some cases, the effect of a one-way valve between the knee joint and the gastrocnemius-semimembranosus bursa can lead to an accumulation of synovial fluid, referred to as a popliteal (Baker) cyst.[4]

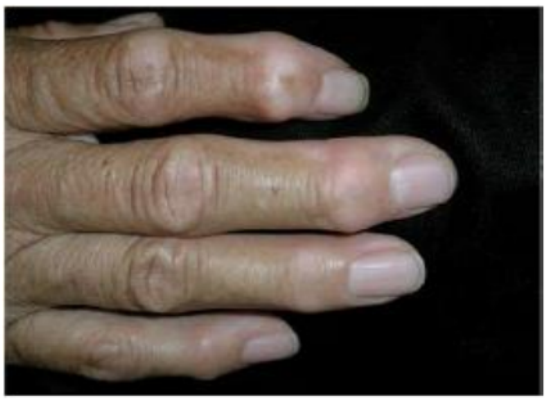

Figure 1. Osteoarthritis in a middle-aged woman with Heberden's nodes in the DIP joint. Swelling at the onset of PIP causes Bouchard's nodes

Investigations in osteoarthritis

Laboratory findings

Osteoarthritis does not cause an increase in the erythrocyte sedimentation rate (ESR) or other laboratory signs of inflammation. Synovial fluid is non-inflammatory.[5]

Imaging

Radiographs may reveal joint space narrowing, osteophyte formation and marginal bone lip, and thickened dense subchondral bone. Bone cysts may also be present.[5]

Differential Diagnosis

Because articular inflammation is minimal and systemic manifestations are absent, degenerative joint disease is rarely confused with other inflammatory joint diseases. The distribution of joint involvement in the hands also helps differentiate osteoarthritis from rheumatoid arthritis. Osteoarthritis primarily affects the DIP and PIP joints and parts of the wrist and metacarpophalangeal (MCP) joints. Rheumatoid arthritis involves the wrist and the MCP and DIP joints. Furthermore, the enlarged joints become hard and cold in osteoarthritis but supple and warm in rheumatoid arthritis. Skeletal symptoms occur due to degenerative changes in the joints, especially in the spine and can lead to metastatic neoplasia.[2]

Prevention of osteoarthritis

Weight loss reduces the risk of developing knee and hand symptoms of osteoarthritis. Correcting a leg length difference of more than 1 cm with modified shoes can prevent knee osteoarthritis from developing in the shorter leg.[6]

Management

General action

Patients with osteoarthritis of the hand can benefit from assistive devices and instructions on joint protection techniques; the belt is useful for them with symptoms of osteoarthritis of the first carpometacarpal joint. Patients with mild to moderate osteoarthritis of the knee or hip should participate in a regular exercise program (e.g., a supervised walking program, hydrotherapy classes) and, if overweight, must lose weight. The use of assistive devices (e.g, a cane on the contralateral side) may improve functional status.[7]

Medicines

Oral nonsteroidal anti-inflammatory drugs (NSAIDs)

NSAIDs are more effective than acetaminophen for osteoarthritis but have greater toxicity. NSAIDs inhibit cyclooxygenase (COX), the enzyme that

converts arachidonic acid to prostaglandins. Prostaglandins play an important role in promoting inflammation, but they also help maintain homeostasis in several organs—especially the stomach, where prostaglandin E functions as a local hormone responsible for gastric mucosal cytoprotection. COX exists in two isomers—COX-1, which is expressed continuously in many cells and is responsible for the homeostatic effects of prostaglandins, and COX-2, which is induced by cytokines and expressed in inflammatory tissues. Most NSAIDs inhibit both isomers. Celecoxib is the only selective COX-2 inhibitor currently available in the United States.[8]

Gastrointestinal toxicity, such as gastric ulceration, perforation, and gastrointestinal bleeding, is the most common serious side effect of NSAIDs. The overall rate of bleeding with NSAID use in the general population is low (1:6000 users or less) but is increased by risk factors for long-term use, higher doses of NSAIDs, corticosteroids or concomitant anticoagulants, presence of rheumatoid arthritis, history of peptic ulcer disease or alcoholism, and age over 70. Proton pump inhibitors and histamine type-2 receptor antagonists reduce the incidence of serious gastrointestinal toxicity and should be used for patients with risk factors for NSAID-induced gastrointestinal toxicity. Patients who have recently recovered from NSAID-induced bleeding gastric ulcer appear to be at high risk for rebleeding (approximately 5% at 6 months) when NSAIDs are reintroduced, even if prophylactic measures (such as proton pump inhibitors) are used. Compared with nonselective NSAIDs, celecoxib is less likely to cause upper gastrointestinal side effects, including bleeding.

All NSAIDs, including aspirin and celecoxib, can produce toxicity, including interstitial nephritis, nephrotic syndrome, prerenal azotemia, and worsening hypertension. Hyperkalemia due to hyporeninemic hypoaldosteronism is rarely seen. The risk of renal toxicity is low but is increased by the following risk factors: age older than 60 years, history of renal disease, heart failure, ascites, and use of diuretics.[9]

All NSAIDs, except nonacetate salicylates and celecoxib, interfere with platelet function and prolong bleeding time. Aspirin irreversibly inhibits platelet function, so the effect of bleeding time is lost only when new platelets are made. In contrast, the effect of nonselective NSAIDs on platelet function is reversible and disappears as the drug is cleared. Concomitant administration of nonselective NSAIDs may impair aspirin's ability to acetylate platelets and thus may interfere with the cardioprotective effects of low-dose aspirin. The US Food and Drug Administration (FDA) has warned that all NSAIDs can increase the risk of myocardial infarction and stroke in patients with or without known risk factors for heart disease or heart disease. While cardiovascular risk is related to dose and duration of treatment, stroke and myocardial infarction can occur within the first week of treatment. The cardiovascular risks associated with moderate-dose naproxen, ibuprofen, and celecoxib (200 mg orally daily) are comparable. Chondroitin sulfate and glucosamine, alone or in combination, were no better than placebo in reducing pain in patients with knee or hip osteoarthritis.

Topical therapy

Topical NSAIDs (e.g, 4 g of 1% diclofenac gel applied to the affected joint four times daily) appear to be more effective than placebo for knee and hand osteoarthritis and has a lower rate of systemic side effects than oral NSAIDs. Topical NSAIDs are preferred for patients 75 years of age and older. Topical capsaicin may be beneficial for hand or knee osteoarthritis.[10]

Acetaminophen and opioids

Acetaminophen is not recommended because it does not reduce pain and has a hepatotoxic effect at high doses. Opioids are generally not suitable for the long-term management of pain due to osteoarthritis.

Intra-articular injection

Many patients with moderately severe knee osteoarthritis who do not respond to NSAIDs receive intra-articular injections of corticosteroids, hyaluronic acid, or platelet-rich plasma. Although each of these can temporarily reduce pain, neither has conclusively produced long-term benefits in reducing pain or maintaining function. A 2-year controlled trial showed that injecting the knee with triamcinolone every 6 months was no more effective than injecting saline in reducing knee pain. The American College of Rheumatology does not recommend corticosteroid injections for osteoarthritis of the hand.[11,12]

Duloxetine

For patients with osteoarthritis of the multiple joints who have not responded to or cannot use NSAIDs, the selective serotonin and norepinephrine reuptake inhibitor duloxetine, 30-60 mg orally daily, may reduce pain. The side effect of nausea occurs in 6-15% of patients.[13]

Surgery

Total hip and knee replacement provides excellent symptomatic and functional improvement when joint involvement severely limits walking or causes pain at rest, especially at night. Arthroscopic surgery for knee osteoarthritis is ineffective.

Conclusion

Osteoarthritis causes can be treated using modification of equipment, such as footwear or canes, or by administration of medication.

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