

REVIEW ARTICLE**Updated Management of Osteoarthritis: A Review**MJ Islam¹, MA Yusuf², MS Hossain³, M Ahmed⁴**Abstract**

The goals of the treatment of osteoarthritis (OA) are to alleviate pain and minimize loss of physical function. To the extent that pain and loss of function are consequences of inflammation, of weakness across the joint, and of laxity and instability, the treatment of OA involves addressing each of these impairments. Comprehensive therapy consists of a multimodality approach including non-pharmacologic and pharmacologic elements. Patients with mild and intermittent symptoms may need only reassurance or non-pharmacologic treatments. Patients with ongoing, disabling pain are likely to need both non-pharmacologic and pharmacotherapy. Treatments for knee OA have been more completely evaluated than those for hip and hand OA or for disease in other joints. Thus, while the principles of treatment are identical for OA in all joints, we shall focus below on the treatment of knee OA, noting specific recommendations for disease in other joints, especially when they differ from those for disease in the knee. [*Journal of Science Foundation, 2013;11(2):49-55*]

Keywords: Osteoarthritis, treatment, pharmacotherapy, cartilage regeneration

[Cited as: Islam MJ, Yusuf MA, Hossain MS, Ahmed M. Updated Management of Osteoarthritis: A Review. J Sci Found, 2013;11(2):49-55]

Introduction

Osteoarthritis (OA) is the most common form of arthritis accounting for about 30% of general physician visits (Kramer et al., 1983). It may be defined as a heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone and at the joint margins (Altman et al., 1986). It is usually classified as either primary (idiopathic) or secondary associated with a known condition. Although OA is present by histologic or radiographic criteria in nearly 80.0% of people by the age of 80 years, only half have symptoms (Hochberg et al., 1989) and these are often variable and intermittent. There is a modest correlation between the presence of symptoms and the severity of anatomic changes.

Clinical Features

All though variable in its presentation and course of OA often carries significant morbidity. In addition to the effects on the individual, the cost of OA to society is significant (Lawrance et al., 1989) related to its high prevalence, the reduced ability of those affected to perform both occupational and non-occupational

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activities, the occasional loss of a patient's ability to undertake self-care, and the related drain on health-care resources (Levy et al., 1993). Although symptoms are often unilateral, evidence of OA is almost always present bilaterally. However, even when symptoms are bilateral, there is a tendency for one side to be more symptomatic than the other. The symptomatic side may also alternate over time. Unilateral disease may suggest OA secondary to trauma. In contrast to systemic inflammatory arthritis, OA lacks constitutional symptoms. When OA is symptomatic, the most prominent complaint is pain. The onset of OA symptomatology is insidious. OA is characterized clinically by pain, swelling of joint and limitation of motion. Pathologically and radiologically the disease is characterized by focal erosive lesions, cartilage destruction, subchondral sclerosis, cyst formation and large osteophyte at the margin of the joints (Mankin 2005).

Pathogenesis

Osteoarthritis is no longer considered 'degenerative' or 'wear and tear' arthritis, rather involves dynamic biomechanical, biochemical and cellular process (Kuettner et al., 1995). Indeed, the joint damage that occurs in OA is, at least in part, the result of active remodeling involving all the joint structures. Although articular cartilage is at the center of change, OA is currently viewed as a disease of the entire joint and therefore, the failure of the joint as an organ (Lozada and Altman 1997). Pain relief is still a primary goal in treating patients who have knee OA. However, pain may have a protective role for the affected knee by causing a reduction in weight bearing (Sater and Field 1960). Therefore, simply alleviating pain may lead to further joint and cartilage damage (Mankin 1974). The development of weakness in muscles that bridge osteoarthritic joints is multifactorial in etiology (Altman and Lozada 2004). First, there is a decline in strength with age. Second, with limited mobility comes disuse muscle atrophy. Third, patients with painful knee or hip OA alter their gait so as to lessen loading across the affected joint, and this further diminishes muscle use. Fourth, "arthrogenous inhibition" may occur, whereby contraction of muscles bridging the joint is inhibited by a nerve afferent feedback loop emanating in a swollen and stretched joint capsule; this prevents maximal attainment of voluntary maximal strength. Since adequate muscle strength and conditioning are critical to joint protection, weakness in a muscle that bridges a diseased joint makes the joint more susceptible to further damage and pain (Wada et al., 1993). The degree of weakness correlates strongly with the severity of joint pain and the degree of physical limitation. One of the cardinal elements of the treatment of OA is to improve the functioning of muscles surrounding the joint (Spector et al., 1997).

Diagnosis

There are no laboratory tests that are diagnostic of OA (Spector et al., 1997). Diagnosis of osteoarthritis is based on clinically and radiologically. X-ray evidence is joint space narrowing, subchondral sclerosis or osteophyte formation and symptoms of pain in the affected knee on motion or rest plus at least one of the following, tenderness with pressure; mild swelling; crepitous on motion; or stiffness, either in morning or after prolonged activity (Fargas-Babjak et al., 1989). Traditionally, OA has been viewed as an inevitable degenerative condition of the cartilage. It is now viewed as a biomechanical and biochemical inflammatory disease of the entire joints (Liang and Fortin 1991). New insights into pathogenesis have revealed a role for inflammatory pathways in the natural history of disease (Poole and Howell 2001). Until a structure modifying agent is available, the objectives in managing the patient with OA knee are reducing/eliminating pain and stiffness, maintain/improve mobility, Optimizing function and hence minimizing disability (Liang and Fortin 1991). So, the goals in management of OA are patient education, individualize therapeutic regimen, treating symptoms, minimizing disability & slowing disease progression (Choudhury et al., 2005).

Basic of Management of OA

Therapeutic approaches include pharmacological like analgesic and NSAID, intra-articular agents, glucosamine, hyaluronic acid and topical capsaicin; another is non-pharmacological like patient education, exercise, personal contact, physiotherapy, assistive device, patellar tapping appropriate footwear and surgical intervention may be needed (Choudhury et al., 2005). In 1995, the American College of Rheumatology (ACR) published guidelines for the treatment of OA knee (Hochberg 1995). These were updated in 2000 (ACR 2000) and 2003 (ACR 2003) and state that, for mild symptomatic OA, treatment may include non-pharmacologic methods patient education, physical & occupational therapy and other therapies

and pharmacologic therapy including non-opioid oral and topical like applied to skin analgesics. For patient who is unresponsive to this regimen, the use of non steroidal antiinflammatory drugs (NSAIDs) is considered appropriately. A corticosteroid injection is recommended for patients with knee OA, particularly when signs of local inflammation with joint effusion are present. Patients with severe symptoms of OA of the knee may require surgical intervention e.g. osteotomy or local joint arthroplasty (Bellamy et al., 2007). Autologous chondrocyte implantation may be a possible treatment. Clinical trials employing tissue-engineering methods have demonstrated regeneration of cartilage in damaged knees, including those that had progressed to osteoarthritis (Hollander et al., 2006). Heat therapy is frequently prescribed to patients with symptomatic knee OA (Giombini et al., 2007). Deep hyperthermia via localized Micro Wave Diathermy (MWD) is effective in several musculoskeletal painful conditions (Giombini et al., 2006).

Non-pharmacotherapy

Since OA is a mechanically driven disease, the mainstay of treatment involves altering loading across the painful joint and improving the function of joint protectors, so they can better distribute load across the joint (Robertson et al., 2005). Ways of lessening focal load across the joint include avoiding activities that overload the joint, as evidenced by their causing pain; improving the strength and conditioning of muscles that bridge the joint, so as to optimize their function; and unloading the joint, either by redistributing load within the joint with a brace or a splint or by unloading the joint during weight bearing with a cane or a crutch. The simplest effective treatment for many patients is to avoid activities that precipitate pain (Bellamy et al., 2007). For the middle-aged patient whose long-distance running brings on symptoms of knee OA, a less demanding form of weight-bearing activity may alleviate all symptoms (Weinberger et al., 1988). For an older person whose daily constitutionals up and down hills bring on knee pain, routing the constitutional away from hills might eliminate symptoms. Each pound of weight increases the loading across the knee three- to six fold. Weight loss may have a commensurate multiplier effect, unloading both knees and hips. Thus, weight loss, especially if substantial, may lessen symptoms of knee and hip OA (Choudhury et al., 2005).

Exercise

Osteoarthritic pain in knees or hips during weight bearing results in lack of activity and poor mobility and, because OA is so common, the inactivity that results represent a public health concern, increasing the risk of cardiovascular disease and of obesity (Cetin et al., 2008). Aerobic capacity is poor in most elders with symptomatic knee OA, worse than others of the same age. At least for knee OA, trials have shown that exercise lessens pain and improves physical function (Bellamy et al., 2007). Most effective exercise regimens consist of aerobic and/or resistance training, the latter of which focuses on strengthening muscles across the joint. Exercises are likely to be effective, especially if they train muscles for the activities a person performs daily. Some exercises may actually increase pain in the joint; these should be avoided, and the regimen needs to be individualized to optimize effectiveness and minimize discomfort. Range-of-motion exercises, which do not strengthen muscles, and isometric exercises that strengthen muscles, but not through range of motion, are unlikely to be effective by themselves. Isokinetic and isotonic strengthening that occurs when a person flexes or extends the knees against resistance have been shown consistently to be efficacious (Kaplan et al., 2003). Low-impact exercises, including water aerobics and water resistance training, are often better tolerated by patients than exercises involving impact loading, such as running or treadmill exercises. A patient should be referred to an exercise class or to a therapist who can create an individualized regimen, and then an individualized home-based regimen can be crafted.

Correction of Mal-alignment

Mal-alignment in the frontal plane (varus-valgus) markedly increases the stress across the joint, which can lead to progression of disease and to pain and disability (ACR 2000). Correcting mal-alignment, either surgically or with bracing, can relieve pain in persons whose knees are maligned. Mal-alignment develops over years as a consequence of gradual anatomic alterations of the joint and bone, and correcting it is often very challenging. One way is with a fitted brace, which takes an often varus osteoarthritic knee and straightens it by putting valgus stress across the knee. Unfortunately, many patients are unwilling to wear a realigning knee brace, plus in patients with obese legs, braces may slip with usage and lose their realigning

effect (Bellamy et al., 2007). They are indicated for willing patients who can learn to put them on correctly and on whom they do not slip. Other ways of correcting mal-alignment across the knee include the use of orthotics in footwear. Unfortunately, while they may have modest effects on knee alignment, trials have heretofore not demonstrated efficacy of a lateral wedge orthotic vs. placebo wedges. Pain from the patella-femoral compartment of the knee can be caused by tilting of the patella or patellar mal-alignment with the patella riding laterally (or less often, medially) in the femoral trochlear groove. Using a brace to realign the patella, or tape to pull the patella back into the trochlear sulcus or reduce its tilt, has been shown, when compared to placebo taping in clinical trials, to lessen patella-femoral pain. However, patients may find it difficult to apply tape, and skin irritation from the tape is common. Commercial patellar braces may be a solution, but they have not been tested. While their effect on mal-alignment is questionable, neoprene sleeves pulled to cover the knee lessen pain and are easy to use and popular among patients. The explanation for their therapeutic effect on pain is unclear. In patients with knee OA, acupuncture produces modest pain relief compared to placebo needles and may be an adjunctive treatment (Uthman et al., 2003).

Pharmacotherapy

While non-pharmacologic approaches to therapy constitute its mainstay, pharmacotherapy serves an important adjunctive role in OA treatment. Available drugs are administered using oral, topical, and intra-articular routes. Acetaminophen, Non-steroidal Anti-Inflammatory Drugs (NSAIDs), and COX-2 Inhibitors Acetaminophen (paracetamol) is the initial analgesic of choice for patients with OA in knee, hip, or hands (Godwin and Dawes 2004). For some patients, it is adequate to control symptoms, in which case more toxic drugs such as NSAIDs can be avoided. Doses up to 1 g 4 times daily can be used (Bellamy et al., 2007). Alternative anti-inflammatory medications are cyclooxygenase-2 (COX-2) inhibitors. While their rate of GI side effects may be less than for conventional NSAIDs, their risk of causing edema and renal insufficiency is similar. In addition, COX-2 inhibitors, especially at high dose, increase the risk of myocardial infarction and of stroke. This is because selective COX-2 inhibitors reduce prostaglandin I₂ production by vascular endothelium, but do not inhibit platelet thromboxane A₂ production, leading to an increased risk of intravascular thrombosis (Uthman et al., 2003).

Intra-articular Injections

Since synovial Inflammation is likely to be a major cause of pain in patients with OA, local anti-inflammatory treatments administered intra-articularly may be effective in ameliorating pain, at least temporarily. Glucocorticoid injections provide such efficacy, but work better than placebo injections for only 1 or 2 weeks. This may be because the disease remains mechanically driven and, when a person begins to use the joint, the loading factors that induce pain return. Glucocorticoid injections are useful to get patients over acute flares of pain and may be especially indicated if the patient has coexistent OA and crystal deposition disease, especially from calcium pyrophosphate dihydrate crystals (Robertson et al., 2005). There is no evidence that repeated glucocorticoid injections into the joint are dangerous. Hyaluronic acid injections can be given for treatment of symptoms in knee and hip OA, but there is controversy as to whether they have efficacy vs. placebo. Optimal therapy for OA is often achieved by trial and error, with each patient having idiosyncratic responses to specific treatments. When medical therapies have failed and the patient has an unacceptable reduction in their quality of life and ongoing pain and disability, then at least for knee and hip OA, total joint arthroplasty is indicated.

Intrarticular (IA) corticosteroid injections have been used for decades in clinical practice for pain relief and control of local inflammation in OA (Dieppe et al., 1993). Intrarticular corticosteroid injections are part of the treatment paradigm suggested in the American College of Rheumatology (ACR) practice for the treatment of knee OA³⁴. However, this practice is still controversial because there is fear that these injections especially when used repeatedly as long term treatment, could promote joint destruction and tissue atrophy. Conversely, studies both in vitro and in vivo in experimental models have shown that corticosteroid injections can, in fact, reduce progression of structural changes.

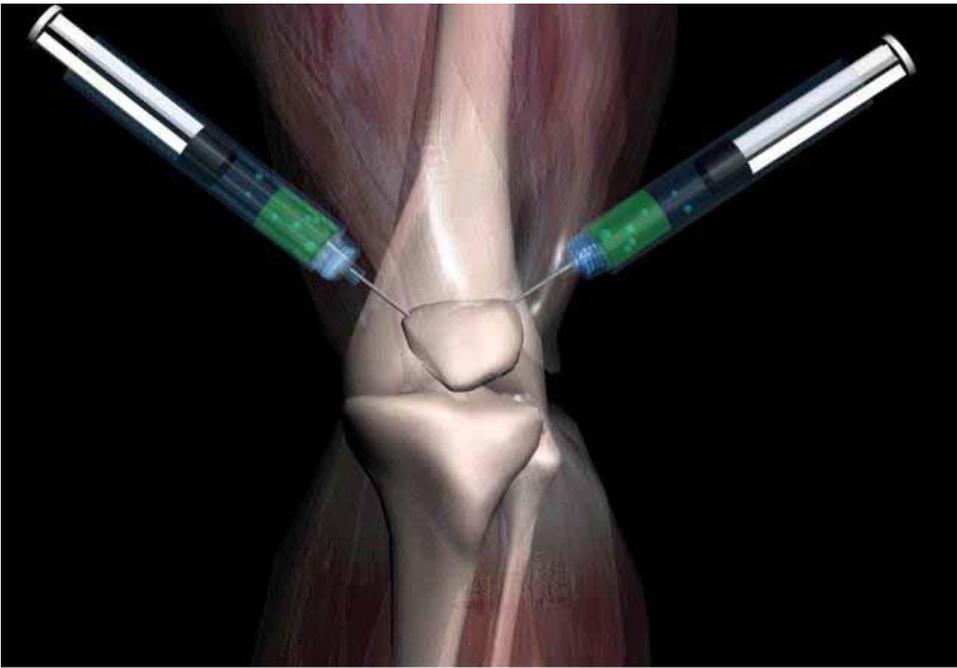


Figure 1: Procedure of intra articular injection (Uthman et al., 2003)

Surgery

For knee OA, several operations are available. Among the most popular surgeries, at least in the United States, is arthroscopic debridement and lavage. A well done randomized trial evaluating this operation showed that its efficacy was no greater than that of sham surgery for relief of pain or disability. There is controversy as to whether mechanical symptoms such as buckling, which are extremely common in patients with knee OA, respond to arthroscopic debridement. While buckling is usually due to muscle weakness, a history of a recent injury, along with knee catching or locking, may suggest a meniscal tear is contributing to this symptom. In such cases arthroscopic debridement with partial meniscal resection might be warranted. For patients with knee OA isolated to the medial compartment, operations to realign the knee to lessen medial loading can relieve pain (Gaffney et al., 1995). These include a high tibial osteotomy, in which the tibia is broken just below the tibial plateau and realigned so as to load the lateral, non-diseased compartment, or a unicompartmental replacement with realignment. Each surgery may provide the patient with years of pain relief before they require a total knee replacement.

Ultimately, when the patient with knee or hip OA has failed medical treatment modalities and remains in pain, with limitations of physical function that compromise the quality of life, the patient should be referred for total knee or hip arthroplasty (Gaffney et al., 1995). These are highly efficacious operations that relieve pain and improve function in the vast majority of patients. Currently failure rates are ~1% per year, although these rates are higher in obese patients. The chance of surgical success is greater in centers where at least 50 such operations are performed yearly or with surgeons who perform a similar number annually. The timing of knee or hip replacement is critical. If the patient suffers for many years until their functional status has declined substantially, with considerable muscle weakness, postoperative functional status may not improve to a level achieved by others who underwent operation earlier in their disease course.

Cartilage Regeneration

Chondrocyte transplantation has not been found to be efficacious in OA, perhaps because OA includes pathology of joint mechanics, which is not corrected by chondrocyte transplants. Similarly, abrasion arthroplasty (chondroplasty) has not been well studied for efficacy in OA, but it produces fibrocartilage in place of damaged hyaline cartilage. Both of these surgical attempts to regenerate and reconstitute articular cartilage may be more likely to be efficacious early in disease when joint mal-alignment and many of the other non-cartilage abnormalities that characterize OA have not yet developed (Ravaud et al., 1999).

Conclusion

Treatment of osteoarthritis is updated from conservative to surgery. Proper treatment is vital need to cure the patients presenting with OA. Appropriate management is the mainstay for the complete recovery of the OA. The associated pathologies should be cured accordingly.

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