CHAPTER 1

CONSERVATIVE TREATMENT OF THE OSTEOARTHRITIC ANKLE

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INTRODUCTION

Osteoarthritis (OA) or degenerative joint disease (DJD) is the most common destructive arthritic diagnosis that affects the ankle joint; the others being (inflammatory) collagen disease, including rheumatoid arthritis, and postinfectious arthritis. The progression of primary osteoarthritis is usually quite slow whereas secondary post-traumatic degenerative arthritis can vary from somewhat slow to surprisingly rapid joint destruction. Other secondary causes include deformities from neuologic disorders such as postpolio, and advanced valgus malalignment from severe and progressive flexible flatfoot or even rigid flatfoot deformity.

OA of the ankle affects 1-4% of the adult population. Its relative low incidence as a primary disease in the ankle, in spite of its small joint surface area and high contact stress as a major weight bearing joint, is quite remarkable. It is thought to be due largely to the biomechanical properties of the joint as it acts as a relatively well-constrained hinge joint with articular cartilage that exhibits a greater capacity for repair. Thus, trauma is the most common cause of ankle osteoarthritis.

Reviewing 639 new patients presenting to their tertiary orthopedic department between 1999 and 2004 with arthritis of the ankle, Saltzman et al (2005) found that 445 (70%) were post-traumatic, 76 (12%) were rheumatoid, and 46 (12%) were idiopathic (primary OA). Post-traumatic arthritic ankles had a strong association with previously treated rotational ankle fractures. Valderrabano et al (2009) looked at 406 ankles with end-stage OA. They observed 78% with post-traumatic arthritis, 13% with secondary arthritis, and 9% with primary OA. Further break-down of the post-traumatic OA ankles, 62% were affiliated with past ankle fractures while 16% had post-traumatic ligamentous OA. Horisberger et al found that malleolar fractures (39%) and tibial plafond fractures were the most common causes of post-traumatic ankle OA. Adequacy of reduction and severity of the injury itself likely play a role in the development of post-traumatic ankle OA, although almost any ankle fracture has the propensity to initiate an OA deterioration of this functionally-important joint, regardless of treatment.

Accelerated cartilage breakdown can also occur in the chronically unstable ankle that has been subjected to a severe or recurrent ligament sprains. Further, traumatically induced osteochondral lesions of the dome of the talus can progress to debilitating OA. The bottom line is that ankle injury, whether fracture or ligament related, leads to malalignment, incongruent joint loading, and asymmetrical wear patterns that destroy various amounts of the articular cartilage. This process leads to functionally-limiting joint pain and disability.

IMPACT OF THE DISEASE

Joint pain and loss of ankle motion can lead to significant disability. In assessing the amount of pain, diminished quality of life, and limited physical function reported by patients with ankle OA, Saltzman et al found these effects to be as severe as those as in patients with hip OA. Other studies that analyzed the self-reported physical function in patients with ankle OA using the Short Form 36 tool, noted it to be equivalent to or worse than that in patients with end-stage kidney disease, congestive heart failure, or cervical spine pain, and radiculopathy. Further observations have indicated that patients with ankle OA are usually younger than those with knee or hip OA.

Patients with end-stage ankle OA exhibit an antalgic protected-loading pattern during gait with aberrant muscle function, joint kinetics, kinematics, and plantar pressures. Such dysfunction results in significant muscle atrophy manifested by reduced isometric plantar flexion torque and diminished cross-sectional muscle area. In the big picture, with these affected patients being younger and having to deal with their reduced quality of life over a longer projected life span, there is obviously a profound adverse effect that ankle OA has on their disability.

Conservative measures seem to work better on patients without large areas of exposed subchondral bone denuded of cartilage and a slow progression of symptoms as reported by Martin et al. Those patients with rapid onset of cartilage degeneration following acute trauma or those with large areas of cartilage loss and joint narrowing tend to respond more poorly to nonsurgical treatment.

EDUCATION AND CARDIOVASCULAR EXERCISE

A discussion of weight loss is important in overweight individuals. Each pound of weight loss results in a 4-fold load reduction at the knee with a corresponding effect on the ankle. Also, avoiding excessive stair use will help avoid putting high loads on the joint.

Most patients with advanced OA of their ankle desire to be quite active, especially the younger their age. Some are used to being very athletic. Therefore, it behooves the health care provider to educate the patient as to the nature of the disease process and the contribution of various stresses to its progression, particularly those that involve impact exercises and cyclical axial loading. Counseling the patient to replace jogging, recreational basketball, and tennis with walking, an elliptical or rowing machine, and swimming not only helps to maintain cardiovascular fitness but can often realize substantial relief of symptoms.

PHYSICAL MEASURES

A good starting point might be simple measures such as ice to the ankle after activity and heat for the chronic pain. Since ankle dorsiflexion and plantarflexion strength have been found to be decreased in the arthritic ankle, strengthening exercises can help stabilize the affected joint. Pain and inflammation lead to joint stiffness so joint mobilization, stretching, and range of motion exercises are indicated. Last, but not least, manual joint distraction can lead to joint mobility and a reduction in symptoms. Although it falls under surgical treatment, distraction arthroplasty using an external fixator has been amazingly helpful, particularly in younger patients with mild arthritis and therefore can be termed a "conservative surgical treatment."

Inserting a heel lift into the shoe can relieve stresses on the anterior ankle joint, thereby improving both function and symptom relief. Sometimes this needs to be balanced with a lift in the contralateral shoe to prevent asymmetrical knee and back strain. The use of a single-point cane can be encouraged as it can off-load 11-25% of the body weight from the extremity.

BRACING

According to Rao et al, orthotic management for ankle OA is aimed at reducing pain by maintaining talar alignment and limiting ankle motion during gait. Rocker sole cast boots and solid-ankle cushion-heel braces decrease ankle motion while walking or climbing stairs, as do custom ankle-foot orthoses (AFOs) and rigid hindfoot orthoses. Articulated AFOs are ineffective at controlling hindfoot motion. Some of the best control of ankle joint motion that can sometimes provide dramatic symptom relief is from the custom-fitted leather ankle gauntlet, also known as the Arizona ankle brace. This can be adjusted for comfort and stiffness.

More cosmetically appealing but less motion-controlling devices are the foot orthoses, which can be used interchangeably in multiple pairs of shoe. They help stabilize the ankle and have shown promising results in treating arthritis of the foot.

TOPICAL TREATMENT

Topical agents, especially those with anti-inflammatory properties that reach the inflamed tissues via the transcutaneous route, help avoid oral medication side-effects. They include the trolamine salicylate preparation, Mobisyl cream (B. F. Ascher & Co.) and the newer 3% diclofenate sodium, Solaraze gel (Doak Dermatologics). Other preparations can be prescribed via compounding pharmacies, e.g., 10% ketoprofen PLO gel.

Topical cryotherapy can be safely recommended for temporary symptomatic relief using such preparations as Biofreeze gel (Performance Health, Inc.) and CryoDerm spray or roll-on solution (Merk Medical Distributors).

ORAL AGENTS

Many nutritional supplements are touted for the treatment of arthritis but few, if any, have been studied as much as the cartilage building blocks, glucsosamine and chondroitin. These products can be taken together or separately as a sulfate salt or as an HCl salt. In spite of the observation that some patients using these preparations for arthritis, particularly of the knee and hip, are convinced that they are beneficial, the evidence to date overwhelmingly fails to conclude that they result in a relevant reduction in joint pain or affect joint space narrowing.

The multi-center, double-blind, placebo-controlled GAIT study randomly assigned 1,583 patients with OA of the knee to different regimens and came to the conclusions above. Further, network meta-analysis of multiple trials of glucosamine and chondroitin treatment of OA further supported the same conclusions.

Most authors agree that neither glucosamine nor chondroitin nor their combination is dangerous. If patients perceive a benefit, there appears to be no potential harm to their taking them as long as the benefit continues and they cover the cost of the preparations themselves. One more study, the Long Term Evaluation of Glucosamine Sulphate Study (LEGS), is more rigidly controlled for the many

variables. However, the first results are scheduled to become available, at the very earliest, in November, 2011.

The nonsteroidal anti-inflammatory drugs (NSAIDs) are proven effective therapies to provide reduction in both the inflammation and pain of arthritis. Patients respond variably to different agents and the medications are safest when used for short terms. But arthritic pain can be relatively unrelenting and patients who benefit from NSAIDs tend to want to use them for long periods, even though they are nonaddictive. Since potential side effects can be quite serious, particularly over time, special attention should be paid to the patient's health history and current conditions before recommending them and their therapy monitored periodically. The safer starting agents are naproxen and ibuprofen with the COX-2 inhibitor, celecoxib, continuing to prove to be relatively safe at its daily therapeutic dose.

Oral prednisone, or its equivalents, are usually reserved to tackle acute and painful inflammatory flares of OA. They are safest when used in short courses of descending doses with the understanding that they must be discontinued when the course is complete. Because of the serious potential side effects, particularly associated with long-term use, low-dose maintenance therapy is rarely used for OA. If so, the therapy must be monitored carefully due to the suppression of the hypothalamic-pituitary-adrenal axis. Also, the oral corticosteroids can produce adverse events if used with NSAIDs, ASA, potassium-depleting drugs, estrogen, and vaccines.

INJECTABLE AGENTS

Viscosupplementation

Intra-articular injection therapy with high molecular weight elastoviscus solutions of hyaluronan or hyalans (cross-linked derivatives of hyaluran) to treat OA is termed viscosupplementation. The function of this treatment is to restore the rheologic properties of synovial fluid.

A major component of articular synovial fluid and the extracellular matrix of hyaline cartilage is the high molecular weight polysaccharide, hyaluronan (HA). Repeating units of glucuronic asid and N-acetylglucosamine, connected by a beta bond, make up this glycosaminaglycan molecule, which is synthesized by the chondrocytes in the cartilage as well as by the fibroblasts of the synovial lining also termed synoviocytes.

The preparation produced for therapeutic injection is hyaluronic acid or, in salt form, sodium hyaluronate and there are 6 derivatives on the market today: Synvisc, Hyalgan, Orthovisc, Supartz, Durolane, and Euflexxa. The first 4 are extracted from rooster combs while Durolane and Euflexxa are bioengineered. There has been some hypotheses that the smaller molecular weight types of hyaluronate, such as that produced as Supartz, are more able to bind to the HA-receptors within the arthritic joint and initiate limited self-production of HA. This is a disease-modifying process that tends to produce a much longer benefit than the buffering effects of normal saline or the temporary anti-inflammatory response to injectable corticsteroids. It has been found to be safe for patient treatment with minimal side-effects.

Viscosupplementation has been found to be an effective alternative to other conservative measures that have failed and for patients who wish to delay or avoid surgery, particularly in the knee. Pleiman et al reviewed the literature up to 2002 and extrapolated that viscosupplementation is reasonable to use in patients with mild to moderate primary OA of the ankle, although they felt that its efficacy would be less for post-traumatic degenerative joint disease. Since then there have been several studies regarding visco-supplementation in the ankle (Salk et al 2006; Sun 2006; Wtteveen et al 2008; Karatosun 2007; and Luciani et al 2008) that have observed significant symptomatic improvement at the 6-month follow-up, extending to 12- and 18-months as well. Even greater improvement has been observed when combined with arthroscopic debridement according to Carpenter and Motley in 2008.

More studies are needed for this therapy especially regarding its promising disease-modifying benefit. The pathophysiology is not well-understood as the hyaluronic acid has been shown to clear from the joint in as little as 1 week; yet the pain relief persists for 6 to 18 months or longer.

Corticosteroid Injections

Intra-articular corticsteroid therapy has been used for arthritic treatment relatively safely for decades. Various preparations are usually combined with local anesthetic agents for both diagnostic and therapeutic purposes. If the patient experiences prompt relief of the joint pain (due to the local anesthetic) then it can be concluded that the degenerated ankle joint itself is the source of their pain. The various steroid compounds are chosen, singly or in combination, based on their solubility, efficacy, and duration of action. They include dexathethasone phosphate, betamthasone phosphate and acetate, methylprednisolone acetate, triamcinolone acetate, and others.

Even though the relief of joint pain can be rather dramatic in most applications, it is usually of relatively short duration with the occasional patient experiencing exceptional long-term improvement. Besides lowering the joint's immuno-defenses against infection somewhat, repeated injections can have deleterious effects on the joint cartilage. However, the most common undesirable

side-effect of corticosteroid injections is the so-called "steroid flare," an intense but self-limiting inflammatory reaction against the foreign material being introduced into the joint. Patient education at the time of the injection can help the patient deal with this temporary discomfort with instructions to treat it symptomatically until it subsides.

Prolotherapy

One other injection treatment that has caught the interest of clinicians at the Mayo clinic and others, deserves attention. Prolotherapy, also known as proliferative injection therapy or regenerative injection therapy, has been used to strengthen tendons and ligaments and reduce joint pain since being introduced to this country from Germany. Its goal is to improve the injected tissue by stimulating tissue growth within the damaged structures. Much of this is accomplished by causing the release of growth factors.

In spite of using various concentrations of irritating or sclerosing solutions to stimulate a localized inflammatory response, prolotherapy, even of joints, has proven to be quite safe. The inflammation results in a wound-healing cascade, which produces the healing new collagen. Injectable agents include but are not limited to 10% dextrose, various concentrations of alcohol, dilutions of phenol, and glycerine. As the various growth factors and their extraction are emerging, direct injection of the growth factors themselves are starting to produce even more promising results.

Two randomized controlled trials by Rabago et al in 2005 on osteoarthritis reported decreased pain, increased range of motion, and increased patellofemoral cartilage thickness after prolotherapy. More clinical and pathophysiologic research is needed for this modality. Unfortunately, a recent study at the Mayo Clinic on the efficacy of prolotherapy for OA of the ankle was discontinued due to failure to enroll an adequate number of patient participants.

CONCLUSION

Surgery for more invasive treatment of painful and debilitating OA of the ankle consists of open and arthroscopic arthroplasty for debridement, distraction arthroplasty with or without debridement, arthrodesis, and total ankle implant arthroplasty. All result in various amounts of success and risks that are substantially higher than conservative care. For example, even though ankle fusion is touted as the "gold standard" for treatment of painful, debilitating end-stage OA of the ankle that has failed conservative care, it is not without potential undesirable consequences. Two long-term studies of this method of treatment (Fuchs et al in 2003 and Buchner and Sabo in 2003) observed degeneration and arthrosis of adjacent joints of the foot, particularly the midtarsal and subtalar joints at an average of 20 and 9.3 years respectively. Therefore, it behooves clinicians to maintain a broad armamentarium of nonsurgical treatments to maintain comfort and function for their patients with ankle OA for as long as possible. The purpose of this review was to present a broad range of these conservative measures along with their benefits, risks, and potential for expected results.

Figure 1A. Oblique view of ankle with severe degenerative joint disease and displacement.

Figure 1B. Lateral view of same ankle demonstrating loss of joint space, displacement and osteophytes due to degenerative joint disease.

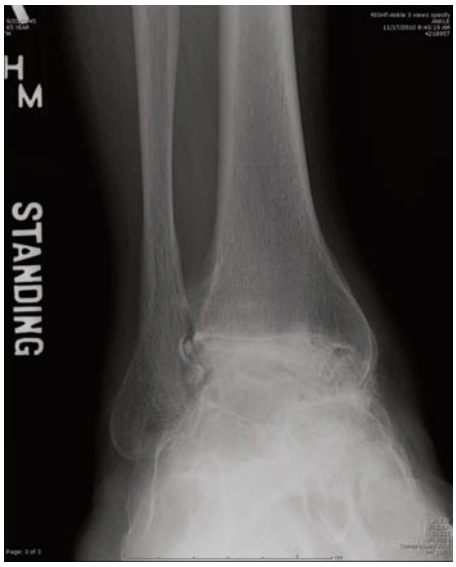

Figure 2A. Mortise view of ankle with end-stage degenerative joint disease as evidenced by complete loss of joint space (photo courtesy of David Calderella, DPM).

Figure 2B. Magnetic resonance imaging of the same ankle showing bone cysts and sclerosis associated with advanced and severe degenerative joint disease (photo courtesy of David Calderella, DPM).

Figure 3A. Talar tilt and sclerotic degenerative joint disease of ankle due to unrepaired posttraumatic lateral ankle sprain (photo courtesy of Tom Brosky, DPM).

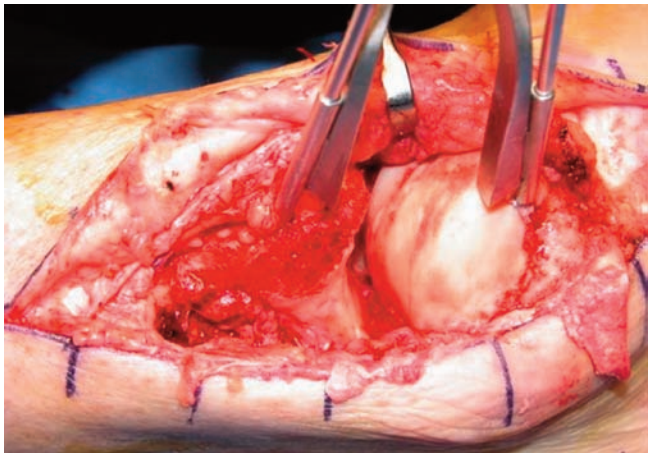

Figure 3B. Surgical exposure of cartilage erosion due to posttraumatic degenerative joint disease in the same ankle. The medial malleolus has been temporarily removed (photo courtesy of Tom Brosky, DPM).