Osteoarthritis: etiology, overview and treatment in both allopathic and functional medicine

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Abstract: Osteoarthritis (OA) is often seen as part of the inevitable aging process and is the most common form of arthritis, affecting an estimated 27 million Americans. 1 This paper will review the etiology of OA, risk factors, current assessment and treatment for OA from an allopathic and functional medicine focus and discuss why reduction of inflammation at a molecular level can reduce OA versus the traditional focus on NSAIDS, along with the connection between metabolic syndrome and OA.

Osteoarthritis (OA) affects an estimated 27 million Americans. 1 Osteoarthritis is a degenerative disease of the cartilage stemming from multiple factors including mechanical stress and biochemical processes resulting in inflammation. This paper will review the etiology of OA, risk factors, current assessment and treatment for OA from an allopathic and functional medicine focus and discuss why reduction of inflammation at a molecular level can reduce OA versus the traditional focus on NSAIDS, along with the connection between metabolic syndrome and OA.

Etiology of OA

Osteoarthritis has long been perceived as "wear and tear" and an inevitable part of the aging process. It can affect one or more of the joints in the body and most commonly affects hands, cervical, thoracic and lumbar spine, hips and knees. In the human body, joints are formed where two bones come together in the joint capsule which is surrounded by the synovial membrane containing the articular cartilage and synovial fluid. Articular cartilage is avascular (containing no blood vessels) impairing its ability to regenerate. Articular cartilage contains chondrocyte cells, which manufacture an extracellular matrix comprised of collagen, glycoproteins, proteoglycans, and hyaluronic acid and is affected by both osmotic and mechanical stress. 3-4 The chondrocytes in the articular cartilage appear to act as sensors and change metabolic activity based on the their cellular environment. Inflammatory markers such as IL-1 (beta) and Tumor Necrosis Factor-alpha increase Nitric Oxide and reactive oxygen species free radicals and cytokine activity, which signal the immune system. The resulting inflammation results in an increased chondrocyte catabolism inhibiting the growth of the extracellular matrix and cell apoptosis, leading to degradation of cartilage and osteophyte formation. 3-4

There are two pathways for OA, bio-mechanical stress and/or systemic inflammation cause the degradation of the chondrocytes which lead to progression of OA. Progression of OA results in progressive reduction of articular cartilage from outer layers through the subarticular cartilage, eventually leading to bone-on-bone articulation and boney overgrowth known as osteophyte formation. 5 The result is pain in joints during movement (and eventually at rest), stiffness, "crackling" (crepitus), poor joint articulation, and swelling. 5 With progression comes increased pain and impact on quality of life, where the affected may have chronic pain, stiffness and be unable to perform their manual job duties or physical activity such as stair climbing, kneeling, squatting, exercise and housework. 1

OA is graded in stages which look at the narrowing of the interarticular space, from early (loss of 10% of cartilage to severe with 60% of cartilage loss). Early OA is reversible if inflammation is resolved. 4,7 Many of the symptoms of OA including osteophyte formation, stiffness from the formation of fibrous growth in joint are related to the bodys attempts at tissue repair stimulated by the increase of cytokines and macrophage activity due to increased inflammation. 4,8

RISK FACTORS

Risk factors for OA include age, with risk increasing annually over the age of 45 (although it can develop in people in their 20's), genetic factors, sex (women are seen as more predisposed to hip OA, which may be related to a broader pelvic structure and also reduction in estrogen levels

following menopause although there is not enough conclusive evidence to support this as of yet). 1-3 Trauma from injury to the joint and overuse/repetitive movement from either job requirements or high-impact exercise can also lead to degradation of cartilage. 1-2. Joint misalignment from increased knee varus (knock knees) or varum (bowlegged), or hip conditions such as legg-calve-perthes disease or increased decreased Q angle in the hip can increase risk of OA due to the increased stress on the affected joint. 3

Obesity is linked with increased risk for OA, due to increased load on weight bearing joints. Interestingly, there is some research showing that leptin may be involved in the onset and development of OA, which links obesity and OA through increased adipocyte factors. This increases inflammatory processes through IL-6 and C-reactive protein and causes catabolization of chondrocytes (which make up cartilage), rather than the previous thinking that obesity is direct cause of OA due to increased weight loading on joints. 3 The result is that people who are obese are at increased risk for issues related to inflammation due to increased leptin.

ASSESSMENT

OA is diagnosed through X-rays to measure the amount of cartilage degradation and formation of osteophytes. There are several questionnaires including the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), which grandes and measures patients self-reporting of impact on activities of daily living and activity limitation based on pain and symptoms. 4 Patients are usually referred to an orthopedic doctor who orders X-rays and possibly blood tests

if Rheumatoid Arthritis is suspected versus OA. Pain location, severity and presence of swelling, redness and other signs of infection are noted and be taken into account. 8

INTERVENTION

The most common intervention for early to mid-stage progression of OA is NSAIDS or prescription medications such as Celebrex, which help reduce pain and inflammation. Joint replacement surgery (mainly total hip arthroplasty or total knee arthroplasty) is performed on patients with late stage OA. Mainstream supplements widely used include Glucosamine and Chondroitin, which have been shown to reduce symptoms in the short term. 6, 8, 10

ALLOPATHIC VS. FUNCTIONAL MEDICINE

Western medicine has traditionally focused on NSAIDS and other medications for reduction of pain and swelling, strengthening of muscles surrounding the affected joint through Physical Therapy intervention and joint-replacement surgery for advanced cases. 8 OA is occasionally treated through education and encouragement for weight-loss in order to reduce strain on weight-bearing joints. Allopathic models generally do not address underlying causes of systemic inflammation.

In contrast, functional medicine looks at both OA caused by both mechanical stress and systemic inflammation, with treatment approach through diet and nutritional supplementation, hopefully along with the addition of physical exercise to strengthen surrounding muscles around the affected joint. Supplementation of long-chain Omega-3 fatty acids containing EPA and DHA

have an anti-inflammatory effect, along with diets that reduce the omega-6/omega-3 fatty acid ratio, dietary changes which incorporate vitamins A, C, D, E and K to promote increased antioxidant and collagen level, and reduction of LDL serum cholesterol. 6-7. There is other research on various nutritional supplements including Boswella serrata, Uncaria spp (cat's claw), Curcumin and Reversatrol. 6-7, 9, 11.

CONCLUSION

Treatment of OA should include a multi-factored, individual approach that incorporates a detailed health history of the patient. Factors to be addressed are the patient's lifestyle, stress levels, physical activity level, and activities of daily living including body mechanics, repetitive movement and stress during both work and recreational activity. The patients' diet and alcohol intake should be reviewed in detail and testing for systemic inflammation should be considered. Does the patient currently have a diagnosis of OA with X-rays and/or a grading of progression done by an orthopedic doctor and review of other interventions previous trialed.

Personally, I do not think that you can make any recommendations for supplements until you know what the patient's current levels are. Looking for systemic inflammation, a practitioner should have results of an extensive blood panel in order to look at levels of Vitamin D, Vitamin C, C-reactive protein, Erythrocyte sedimentation rate (ESR) and possibly hemoglobin A1-C if the person is obese and/or there are concerns interrelated to type 2 diabetes.

Related to the earlier discussion of obesity, increased leptin levels from adipose tissue and increased risk for OA, there is research suggesting that the presence of obesity, diabetes and metabolic syndrome can influence development of OA as a result of metabolic activity created by adipokines, hyperglycemia and endocrine imbalance. 3 In this case, there should be treatment for the underlying causes of the OA for metabolic syndrome and regulation of blood sugar. I feel that often in western medicine this factor is overlooked, and patients are sent for a joint replacement to cure the joint degradation but these other issues, which are major factors in a person's health, are dismissed or treated with medication.

Overall, in early stages of OA, treatment focus on reducing systemic inflammation based on current vitamin levels, eating whole-foods diet, and reduction of refined carbohydrates, artificial sweeteners, and processed foods. It may be a good idea to supplement with long-chain omega-3 fatty acids and possibly other antioxidants based on the person's individual profile and levels. Vitamin D, C and K are all related to the immune system (vit D), collagen formation (vit C) and also to bone health (vit D and vit K) and influence reduction of oxidative stress and inflammation. 7 Research on Vitamin D showed only incidental correlation to OA, however Vitamin D depletion is widespread and mentioned as a general marker of poor health, inflammation and decreased immune system function. 7. There is also some interesting research that Bowellia serrata and Curcumin used together are effective and seem to work in a synergistic fashion when taken together in terms of reducing inflammation and reducing chondrocyte catabolism and cartilage degradation. 11

The final piece of treatment required is exercise and movement. Because OA results in joint pain and stiffness, people often reduce their activity levels, leading to muscle loss (sarcopenia). Gentle movement and strengthening of the muscles surrounding the joint has been shown to improve overall health and maintain strength levels required to maintain functional mobility. 8

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