Review Article

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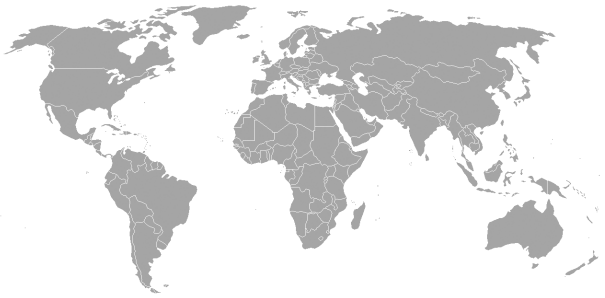
Ostoearthritis Prevention



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ABSTRACT

Osteoarthritis is the most common chronic joint disease causing substantial pain, functional limitations and disability in the world. Although several factors such as mechanic stress, biochemical and genetic factors are involved in the etiology and development of this condition, the exact cause is unknown. Osteoarthritis is among the major causes of disability. Impaired ability to perform daily activities such as walking, ascending or descending stairs, sitting and standing due to hip and knee osteoarthritis occurs more often than those caused by other chronic diseases. Osteoarthritis affects more than half of persons over the age of 65 years and 80% of those older than 75 years old. As the population ages, osteoarthritis will become one of the most important health-care challenges of the future. Since there is no cure for osteoarthritis, prevention and reducing the risk factors are very important. In this review, we will address awareness of the risk factors and some prevention methods.

Key words: *Osteoarthritis, prevention, nutraceuticals*

Osteoartritin Önlenmesi

ÖZET

Osteoartrit en sık rastlanan kronik eklem hastalığıdır. Önemli derecede ağrı, fonksiyonel kısıtlılık ve sakatlığa neden olur. Etiyolojide mekanik stress, biyokimyasal ve genetik faktörler suçlansa da sebebi tam olarak bilinmemektedir. Osteoartrit en sık sakatlık nedenleri arasında yer alır. Kalça ve diz osteoartritinde yürüme, merdiven inip çıkma, oturma ve kalkma gibi günlük yaşam aktivitelerini gerçekleştirmeki kayıp diğer kronik hastalıkların neden olduğundan daha fazladır. Altmış beş yaş üzeri nüfusun yarıdan fazlası, 75 yaş üzerindekilerin ise % 80'I osteoartritten etkilenir. Gelecekte nüfus yaşlandıkça osteoartrit en önemli sağlık sorunlarından biri olacaktır. Kesin bir tedavisi olmadığı için hastalığın önlenmesi ve risk faktörlerinin azaltılması önem arz etmektedir. Bu derlemede risk faktörlerinin farkına varılması ve bazı önleyici yaklaşımlar üzerinde durulacaktır.

Anahtar kelimeler: *Osteoartrit, önleme, besin destekleri*

INTRODUCTION

Osteoarthritis (OA) is the most common chronic joint disease causing substantial pain, functional limitations and disability in the world. It is defined by focal lesions of articular cartilage, combined with a hypertrophic rea ction (sclerosis) in the subchondral bone and new bone formation (osteophytes) at the joint margins (Figure 1). More recently, OA has been relabelled as a whole organ disease, because pathological abnormalities in all the joint tissues, including hyaline cartilage, synovial membrane, menisci, ligaments and subchondral bone and neurosensory system alteration are often present in these patients (1). Although several factors such as mechanic stress, biochemical and genetic factors are involved in the etiology and development of this condition, the exact cause is

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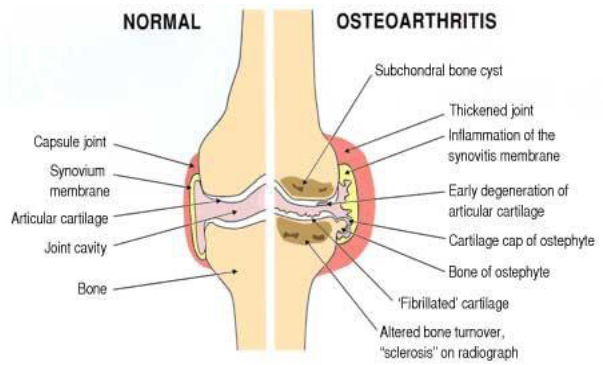

***Figure 1**. Pathology of osteoarthritis*

unknown. Previously, OA was thought to be caused largely by excessive wear and tear, but increasing evidence points to the contributions of abnormal mechanics and inflammation. OA affects more than half of persons over the age of 65 years and 80% of those older than 75 years old (2,3). The advancements in medical science and increased life expectancy indicate that the prevelance of OA will increase in the following years.

Osteoarthritis predominantly involves the weight-bearing joints, including the knees, hips, cervical and lumbosacral spine, and feet. Other commonly affected joints include the distal interphalangeal (DIP), proximal interphalangeal (PIP), and carpometacarpal (CMC) joints (Figure 2). Clinically OA is characterised by joint pain, crepitus, stiffness and limitation of movement. OA is among the major causes of disability (4). Loss of mobility and impaired ability to perform daily activities such as walking, ascending or descending stairs, sitting and standing due to hip and knee OA occurs more often than those caused by other chronic diseases (5). As the population ages, OA will become one of the most important health-care challenges of the future (6). Since there is no cure for OA, reducing the risk factors and prevention methods has utmost importance. In this review, we will address awareness of the risk factors and some preventive methods which can affect the prognosis of the disease. It should not be forgotten that "an ounce of prevention is worth a pound of cure!"

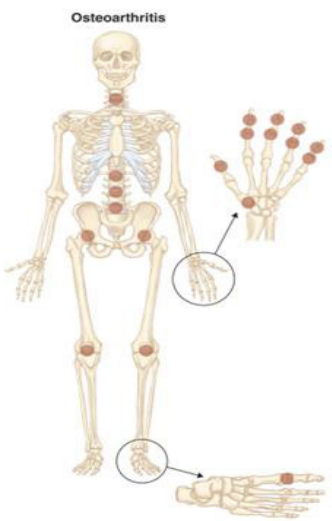

***Figure 2**. Joints involved in osteoarthritis*

Education

Since OA is a disorder that requires adaptation and/or lifestyle changes due to the disease-associated loss or limitation of functions, when the patients are informed regarding these issues, it is easier for them to let go of their negative beliefs associated with their condition, to control their complaints and handle their problems, and their treatment compliance increases. Informing and educating the patients and providing them social support are considered to be parts of the treatment. Tools such as seminars, books, periodicals, brochures, and videos can be beneficial in patient education. Patient education is proved to positively support non-steroidal anti-inflammatory drug (NSAID) treatments by 20-30% (7).

The patients can be informed about the general joint structure, what happens in case of OA, differences between OA-free and OA-affected joints, treatment options, and the required changes to be implemented in their daily lives. Identifying the factors that cause overloading of the joint.

It is effective to educate patients on joint protection in order to prevent both the future pain and the possible future joint damage. The patient should be informed about the activities that overload the stiff joint and the ways to avoid them. Joint use during occupational and non-occupational activities should be evaluated thoroughly. Patients with hip or knee OA should accommodate their work environment in order to refrain from standing for long periods of time and kneeling down.

Exercise

It has been well known that weakness in the muscles stabilizing the joint is associated with the increased risk of OA. Isometric strengthening and closed chain exercises and improving the flexibility are safe and effective for all ages. The another safest exercises are those that place the least body weight on the joints, such as bicycling, swimming and water exercise.

Weight Loss and Other Dietary Recommendations

An epidemiologic association is found between OA and obesity, especially in case of knee OA among women (8). Being overweight in earlier ages has been found to be a strong risk factor for OA development in knees (9). The relative risk factor for knee OA development among individuals with body mass index (BMI) close to the upper limits has been found to be 1.5 among men and 2.1 among women (10). Individuals without OA can reduce their risk of developing OA significantly by not gaining weight or losing weight if they are already overweight. Even a weight loss of only 5 kg can reduce symptomatic knee OA risk by 50% (11). A study has shown that dietary restriction has reduced weight by 28% and the associated pathological severity of the disease by 40% among subjects with knee OA (12). Obese patients can be given dietary prescriptions. However, the immobility due to OA and inability to adapt to a low-calorie diet hinders weight loss.

Dietary Supplements: «Nutraceuticals»

Many products classified as dietary supplements category that are thought to have medical effects, especially glucosamine and chondroitin sulfate, are in high demand in OA treatment (13). Glucosamine is an amino-saccharide that is found in the structures of cartilage proteoglycans and hyaluronic acid. The dose of 1500 mg/day of glucosamine sulfate is reported to positively affect OA complaints and to prevent articular cartilage damage (14). Another study on glucosamine sulfate report that health related disability scores are better to those of a placebo, but worse than those of NSAID use (15). Glucosamine sulfate use of 1-3 years is observed to prevent to undergo for a total joint arthroplasty in the following 5 years (16). Nevertheless, a recently Cochrane review has failed to demonstrate glucosamine use on OA over the WOMAC index (17).

A meta-analysis including 3486 knee and hip OA patients has evaluated the chondroitin effectiveness and revealed that the benefits were minimal (18). Another meta-analysis cited by Hochberg confirmed that joint space narrowing is delayed significantly by chondroitin sulfate use (19). Effect of dietary supplements on OA was investigated in a multicenter, randomized, placebo-controlled Glucosamine/Chondroitin Arthritis Intervention Trial (GAIT). A total of 1583 patients with a mean age of 59 years, who were suffering from knee pain and had evidence of OA in direct X-ray, were treated in 5 different groups. The first group was administered 1500 mg of glucosamine only, second group was administered 1200 mg of chondroitin sulfate only, third group was administered glucosamine and chondroitin sulfate in combination, fourth group was administered placebo, and the fifth group was administered 200 mg of ibuprofen only (positive control). While no significant benefit of using glucosamine or chondroitin sulfate alone, or their combination could be detected compared to placebo, the combined therapy was observed to lead to a significant decline in the VAS score of patients with moderate pain. No significant difference was found in terms of side effects either (20).

Though studies are limited, s-adenosylmethionine (SAM-e), methylsulfonylmethane (MSM), ginger extracts, soy and various cartilage extracts are also of interest. S-adenosylmethionine (SAM-e) is obtained through methionine digestion in the liver. By blocking the cytokine expression, it prevents chondrocyte damage and retains the joint cartilage thickness. Methylsulfonylmethane (MSM) is oxidized from dimethyl sulfoxide (DMSO) in adrenal glands. In a meta-analysis of 11 randomized controlled trials comparing SAM-e and NSAID use, it was shown that SAM-e appears to be as effective as NSAIDs in reducing pain and improving functional limitation in patients with OA without the adverse effects often associated with NSAID therapies (21).

MSM use at 500 mg 3x1/day dose alone and in combination with glucosamine hydrochloride at a dose of 500 mg 3x1/day were observed to improve Likert scale and Lequesne index values (22). MSM use at a dose of 3 gr 2x1/day was shown to cause a significant decrease in the WOMAC pain and function score when compared to placebo (23). In-vitro and animal trials have confirmed that fish oil preparates including polyunsaturated fatty acids (PUFA) and omega-3 have anti-inflammatory and chondroprotective effects. They are thought to bind to G protein receptors and act as potent inflammation antagonists. Studies on combined PUFA-omega-3 and 1500 mg/day glucosamine sulfate use gave successful results on WOMAC pain/func-

tion development scores (24). A similar randomized controlled study has shown that a fish oil derivative known as "Phytalgic" has positive effects on pain (25).

Avocado/soybean unsaponifiable (ASU) products have both antioxidant and analgesic effects. These effects are provided by blocking the tumor necrosing factor (TNF)-α, interleukin (IL)-1β, inducible nitric oxide synthase (iNOS), cyclooxygenase (COX)-2 products, and prostaglandin (PG)-E2 secretion. A meta-analysis of 4 randomized controlled trials includes 6-month follow-up data on 664 hip and knee OA patients on 300 mg of ASU or placebo. ASU use was found to be significantly superior to placebo in terms of pain management (26). In another meta-analysis (Cochrane) on ASU has shown that it has positive effects on various factors such as functional index, pain, reduced NSAID use, and experiencing fewer side effects (27).

Allium compounds

In a study by Williams et al. it has been shown that the organosulfides, or allyl sulfides compounds known as alliums that are found in garlic, onions, and leeks, actually inhibite the enzymes that cause joints to degenerate(28).

Orthoses, Shoes, and Walking Devices

It is very important to wear appropriate and comfortable shoes, using soft insoles, and walking on appropriate grounds to manage the load on the joints. Using of heel wedges is reported to reduce the effects associated with varus and valgus deformities in mild knee OA. Wedges with 5-10° elevation in the frontal cross-section are proven to benefit the medial OA of the knee (29). The wedge reduces the loading on the medial joint of the knee and the tension on the medial collateral ligament. This condition is accepted as a type of "medical osteotomy."

Simple walking devices such as a cane or a walker have positive impacts on mobility and independent living by reducing the joint overloading. Properly used canes widen the support base and share the load and the overall task of the joint. The sole use of cane can reduce the reaction force on an OA-affected hip by 50% (30). However, most of the OA or at-risk patients are not psychologically prepared to use a device for walking support. To help the patients adapt to this lifestyle change, they should be trained on the mechanical advantages the device will provide, test using the device to see the reduction in pain, and be told that the device will prevent disease progression.

A basic knee pad use can be helpful among knee OA patients with instability or a sensation of their "knee giving out" (31). A Swedish knee cage orthosis or a hinged knee orthosis used to manage the pain and instability of the knee can be beneficial in pain reduction by limiting extension (32). Three-point pressure system orthoses can be used in managing medial and lateral instability. Orthoses are generally not used in case of hip OA, use of a cane on the healthy side is recommended instead. Patellar bandaging is recommended in case of patellofemoral OA to provide the optimal positioning of the patella, unload the pain region, and to relax the quadriceps. Properly applied bandaging can significantly reduce the frontal knee pain (33).

REFERENCES

1. *Felson DT: The epidemiology of osteoarthritis. The results from the Framingham Osteoarthritis Study. Semin Arthritis Rheum 1990;20:42.*
2. *Kallman DA, Wigley FM, Scott WW. New radiographic grading scales for ostoearthritis of the hand: reliability for determining prevalance and progression. Arthritis Rheum 1989; 32:1584-591.*
3. *World Health Organisation and the Bone and Joint Decade 2001. Available at http://www.boneandjointdecade.org*
4. *Superio-Cabuslay E, Ward MM, Orig KR. Patient education interventions in osteoarthritis and rheumatoid arthritis: a meta-analytic comprarison with nonsteroidal antiinflammatory drug treathment. Arthritis Care Res 1996;9:292-301.*
5. *Hochberg MC, Lethbridge-Cejku M, Scott WW Jr. The association of body weight, fatness and body fat distribution with osteoarthritis of the knee: J Rheumatol 1995;22:488-93.*
6. *Felson DT, Anderson JJ, Naimack A. Obesity and symptomatic knee osteoarthritis: Results from the Framingham Study. Arthritis Rheum 1987;30:S130.*
7. *Bray GA, Jordan HA, Sims EA. Evaluation of the obese patient: An algorithm. JAMA 1976; 235:1487-91.*
8. *Felson DT, Zhang Y, Anthony JM. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. Ann Intern Med 1992;117:535.*
9. *Bendele AM, Hulman JF. Effects of body weight restriction on the development and progression of spontaneous osteoarthritis in guinea pigs. Arthritis Rheum 1991;34: 1180.*
10. *Mazzuca SA, Brandt KD, Lane KA. Knee pain reduces joint space width in conventional standing anteroposterior radiographs of osteoarthritic knees. Arthritis Rheum 2002;46:1223-37.*
11. *Reginster J, Derosity R, Rovati L. Long term effects of glucosamine sulphate on osteoarthritis progression. Lancer 2001;357:252-6*
12. *Herrero-Beaumont G, Ivorra JA, Del Carmen TM, et al. Glucosamine sulfate in the treatment of knee osteoar-*

thritis symptoms: a randomized, double-blind, placebo-controlled study using acetaminophen as a side comparator. Arthritis Rheum 2007;56(2):555-67.

13. Bruyere O, Pavelka K, Rovati LC, et al. Total joint replacement after glucosamine sulphate treatment in knee osteoarthritis: results of a mean 8-year observation of patients from two previous 3-year, randomised, placebo-controlled trials. Osteoarthritis Cartilage 2008;16(2), 254-60.

14. Towheed TE, Maxwell L, Anastassiades TP, et al. Glucosamine therapy for treating osteoarthritis. Cochrane Database Syst Rev 2005;2:CD002946.

15. Reichenbach S, Sterchi R, Scherer M, et al. Metaanalysis: chondroitin for osteoarthritis of the knee or hip. Ann Intern Med 2007;146(8):580-90.

16. Hochberg MC. Structure-modifying effects of chondroitin sulfate in knee osteoarthritis: an updated metaanalysis of randomized placebo-controlled trials of 2-year duration. Osteoarthritis Cartilage 2010;18(Suppl 1):S28-31.

17. Sawitzke AD, Shi H, Finco MF, et al. Clinical efficacy and safety of glucosamine, chondroitin sulphate, their combination, celecoxib or placebo taken to treat osteoarthritis of the knee: 2-year results from GAIT. Ann Rheum Dis 2010;69(8).1459-64.

18. Soeken KL, Lee WL, Bausell RB, Agelli M, Berman BM. Safety and efficacy of S-adenosylmethionine (SAMe) for osteoarthritis. J Fam Pract 2002;51(5):425-30.

19. Usha PR, Naidu MU. Randomised, Double-Blind, Parallel, Placebo-Controlled Study of Oral Glucosamine, Methylsulfonylmethane and their Combination in Osteoarthritis. Clin Drug Investig 2004;24(6):353-63.

20. Kim LS, Axelrod LJ, Howard P, Buratovich N, Waters RF. Efficacy of methylsulfonylmethane (MSM) in osteoarthritis pain of the knee: a pilot clinical trial. Osteoarthritis Cartilage 2006;14(3).286-94.

21. Gruenwald J, Petzold E, Busch R, Petzold HP, Graubaum HJ. Effect of glucosamine sulfate with or without omega-3 fatty acids in patients with osteoarthritis. Adv Ther 2009;26(9):858-71.

22. Christensen R, Bliddal H. Is Phytalgic(R) a goldmine for osteoarthritis patients or is there something fishy about this nutraceutical? A summary of findings and risk-of-bias assessment Arthritis Res Ther 12010;2(1):105.

23. Christensen R, Bartels EM, Astrup A, Bliddal H. Symptomatic efficacy of avocado-soybean unsaponifiables (ASU) in osteoarthritis (OA) patients: a meta-analysis of randomized controlled trials. Osteoarthritis Cartilage 2008;16(4):399-408.

24. Little CV, Parsons T. Herbal therapy for treating osteoarthritis. Cochrane Database Syst Rev 2001;(1):CD002947.

25. Yasuda K, Sasaki T. The mechanics of treathment of the osteoarthritic knee with a wedged insole. Clin Orthop 1985; 215:162.

26. Neumann DA. Biomechanical analysis of selected principles of hip joint protection. Arthritis Care Res 1980;80:163-68.

27. Kirkley A. The effect of bracing on varus gonarthrosis. J Bone Joint Surg 1999;80:163-68.

28. Hicks JE, Gerber LH. Rehabilitation in the management of patient with osteoarthritis. Moskowitz R, Howell D, Goldberg V, Mankin H (Ed.): Osteoarthritis: Diagnosis and medical/surgical management. WB Saunders, Philedelphia, 1992, S.427-64

29. Cushnaghan J, McCarthy C, Dieppe P. Taping the patella medially: A new treatment for ostearthritis of the knee joint? BMJ 1994; 318:753.

30. Bateni H, Maki BE. Assistive devices for balance and mobility: benefits, demands, and adverse consequences. Arch Phys Med Rehabil 2005;86:134-45.

31. Dan K R, Mary ER. Unloader Braces for Medial Compartment Knee Osteoarthritis. Sports Health. 2009;1:416-26.

32. Hennessey WJ. Lower limb orthosis. In: Braddom RL, editor. Physical Medicine and Rehabilitation 3rd ed. Philadelphia: Elsevier Inc; 2007. p.343-67.

33. Hassan BS, Mockett S, Doherty M. Influence of elastic bandage on knee pain, proprioception, and postural sway in subjects with knee osteoarthritis. Ann Rheum Dis 2002;61:24-28.