Human Journals

Review Article
February 2021 Vol.:20, Issue:3

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Osteoarthritis: An Overview



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Submitted: 03 January 2021
Revised: 23 January 2021
Accepted: 12 February 2021



www.ijppr.humanjournals.com

Keywords: Osteoarthritis, Pathogenesis, Management

ABSTRACT

Osteoarthritis (OA) is a chronic degenerative disorder of the aged population leading to the destruction of articular cartilage and joints which causes disability. Age, gender, bone density obesity are major risk factors for knee OA. Early joint tissue changes are associated with symptoms like joint agony, joint flimsiness, and solidness. OA is the disease of the whole joint that involves many pathophysiological processes that arise from a dysregulation in the function of cytokines and growth factors, prostaglandins, cartilage matrix fragments, neuropeptides, reactive oxygen intermediates, proteolytic enzymes, and protease inhibitors. Although many treatment plans have been implemented, from the least intense (analgesics) to the most intense (knee replacement [TKR] surgery), it has remained unclear which treatment or combinations of treatments are most effective for the relief of pain and inflammation, improvement and maintenance of mobility, function and health-related quality of life.

INTRODUCTION:

Osteoarthritis is the most common form of rheumatic musculoskeletal disorder. It can affect

any joint, but specifically the knee, hands, hip, and spine with an associated risk of mobility

disability^[1,2].

Osteoarthritis (OA) is portrayed by a degeneration of the articular ligament, during which the

breakdown prompts grid fibrillation, gap appearance, net ulceration, and full-thickness loss of

the joint surface. This is often joined by hypertrophic bone changes with osteophyte

improvement and sub-chondral bone plate thickening^[3].

EPIDEMIOLOGY

It has been conjecture that 25% of the grown-up populace or more than 50 million individuals

within the US are influenced by this illness constantly in 2020 and going to be a big reason

for grimness and physical constraint among people beyond 40 years^[4,5].

The age normalized pervasiveness of radiographic knee OA in grown-ups age is ≥ 45 .The

predominance of OA in any case, shifts enormously relying upon the definition utilized, age,

sex and geological region studied^[6]. The occurrence of hand, hip, and knee OA increments

with age, and ladies have higher rates than men, especially after the age of fifty years. A

levelling off or decay happens in the least joint destinations around the age of 80 years^[7].

RISK FACTORS

Age

The association between age and the risk of OA is probably multifactorial, as a result of

numerous individual factors including oxidative damage, thinning of cartilage, muscle

weakening, and a reduction in proprioception^[8].

Gender

The occurrence of hand, hip, and knee OA is higher in women than men because it increases

drastically around the time of menopause^[9].

Sex hormones

The accelerated age-related rise in OA incidence in women following menopause indicates

the possible role for sex hormones, particularly oestrogen deficiency, in the systemic

predisposition to OA^[10].

Bone density and osteoporosis

People with increased subchondral bone density may be at higher risk for osteoarthritis than

people with less subchondral bone density, because of the stiffer bone's transmitting

mechanical stress to the joints and consequently damaging the articular cartilage^[11].

Nutrition

A role for nutritional factors in OA is recommended by the hypothesis that OA vulnerability

is increased from oxidative damage to cartilage and other joint tissues caused by oxygen

radicals that are produced by chondrocytes in damaged cartilage^[12,13].

Obesity

The relationship between obesity and OA is probable to involve in the effect of excess weight

on overloading of the hip and knee joints throughout the weight-bearing activities, causing

the breakdown of cartilage and damage to ligaments^[14].

Injury/surgery

Acute joint injuries including fractures and dislocations, meniscal and cruciate ligament tears

in the knee, and confer a greater risk of later development of OA in the injured joint^[15].

Physical activity/sports

The risk of OA increases with joint damage and meniscal injury, while ligamentous damage

is continued during sports participation^[16].

SYMPTOMS

Essential side effects of OA incorporate joint agony, joint flimsiness, solidness, radiographic

joint space narrowing, and constraint of development. Sickness movement is generally slow

however can eventually cause joint disappointment with torment and inability.

OA is often characterized as radiological, clinical or emotional. There are numerous endeavors to exactly distinguish and grade radiographic malady in OA. It is most broadly evaluated by utilizing the Kellgren and Lawrence (K&L) score. The overall evaluations of the seriousness are resolved from 0 to 4 and are identified with the assumed consecutive appearance of osteophytes, joint space misfortune, sclerosis, and cysts^[17].

BIOCHEMICAL MARKERS IN OSTEOARTHRITIS

The current finding of osteoarthritis relies upon clinical history and radiography. Radiographic changes happen late within the malady and are generally irreversible. Atomic markers may hypothetically have the choice to spot osteoarthritic changes at a beginning time. During a perfect world, these markers would be touchy to vary, solid, and quantitative^[18].

There are presently a couple of contenders for biochemical markers in osteoarthritis, yet none are seen as explicit up so far. They reflect rebuilding of the bone, ligament, and synovium. Cartilage oligomeric grid protein (COMP) could be a marker of ligament demolition. Cresponsive protein, hyaluronan, YKL-40, and metalloproteases are markers of synovial aggravation^[19].

HUMAN

PATHOGENESIS

The progression of osteoarthritis has been considered as the result of age or trauma which is generally divided into three stages. In the first stage, the proteolytic breakdown of the cartilage matrix occurs which results in loss of compressive resistance of the tissue as selective degradation of collagen leading to the loss of tensile strength^[20,21]. Articular cartilage degeneration is followed by the genesis of new extra bone on trabeculae in the subchondral bone, which may include subchondral sclerosis, growth of osteophytes, the formation of cyst-like bone cavities ^[22]. In the second stage, the erosion of cartilage surface and fibrillation is accompanied by the release of breakdown products into the synovial fluid^[20]. The synovial membrane of osteoarthritic joints is commonly accompanied by focal infiltration of lymphocytes and monocytes in sublining layers^[23]. In the third stage, the synovial inflammation begins when synovial cells and osteoarthritic chondrocytes produce a large number of matrix metalloproteinases (MMPs), like MMP-1, MMP-3, MMP-9, and MMP-13^[24]. It also secretes pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α), which mediate the progression of pain associated with the disease^[25]. Increased expression levels of

osteopontin have been correlated with disease severity which is expressed in high quantity by synovial tissue in osteoarthritis ^[26]. Meniscal tears can be a preceding feature of incipient osteoarthritis, and meniscus damage while extrusion generally has an important role in the structural progression of the disease^[27].

Treatment

Treatment decisions fall into four principle categories: non-pharmacologic, pharmacologic, correlative and elective, and surgical. By and large, treatment should start with the most secure and least obtrusive treatments before continuing to increasingly intrusive, costly treatments. All patients with osteoarthritis ought to get probably some treatment from the initial two classes. Careful management ought to be held for the individuals who don't improve with social and pharmacologic treatment, and who have immovable torment and loss of capacity [28.29].

NON-PHARMACOLOGIC

Physiotherapies are the widely performed non-pharmacological treatment.

The researchers had discovered a genuine critical enhancement in an approved joint pain, as a result of an activity-based program comprised of muscle reinforcing and scope of movement workouts in patient's knee osteoarthritis^[30].

Other than using the mechanical or practical upgrades, simple physical movements or exercises seem to be more advantageous to the patient populace. Also, they tend to offer a probable decrease in diabetes, cardiovascular risks, falls, inability, perking up, and self adequacy.

For patients who are not willing for land-based activity, water-based or aquatic treatments provide solace with a lesser joint effect. Some patients can endure water based therapies and show a reduction in the intensification of symptoms. But they experience some difficulties when starting the weight-bearing sessions.

Some physicians utilize this method of treatment to prolong the therapy in order to give the patients an opportunity for land based modalities when they have lost the apprehension of moving^[31].

PHARMACOLOGIC TREATMENT

CYCLOOXYGENASE INHIBITORS (ACETAMINOPHEN AND NSAIDS)

Acetaminophen, the most regularly utilized drug has demonstrated to be mediocre compared to other NSAIDs and not better than fake treatment for torment control, prompting a few rules to decline to suggest it as a successful clinical administration methodology for moderate-to-extreme OA^[32]. Commonly used NSAIDS are diclofenac, ketoprofen, ibubrufen, acetylsalicylic acid and indomethacin. NSAIDs exert their activities by restraining enzymatic movement of the COX enzymes.

INTERVENTIONAL MANAGEMENT

Multiple substances conveyed through intra-articular (IA) infusions have been investigated previously. The thought behind this is nearby medicines will have less fundamental unfriendly impacts and saving the prescription inside the joint will have a more straightforward impact. Intra-articular corticosteroids are operators diminish cytokine diapedesis inside the slender endothelium, repress incendiary cell collection, attachment, phagocytosis and immunoglobulin blend just as the arrival of prostaglandin and leukotriene. Visco supplementation with HA subsidiaries essential instrument of activity is the reclamation of the lubrication of the joint.

CORTICOID INFUSIONS

Corticoids (CS), inspire their immunosuppressive and mitigating impacts by acting legitimately on atomic receptors, interfering with the provocative course at numerous levels. They decline the activity and creation of IL-1, leukotrienes, prostaglandins, and metalloproteinases. Corticosteroids utilized for the treatment of knee osteoarthritis incorporate triamcinolone acetonide, methylprednisolone acetic acid derivation, betamethasone acetic acid derivation, betamethasone sodium phosphate, and dexamethasone sodium phosphate.

VISCOSUPPLEMENTATION WITH HYALURONIC CORROSIVE

Hyaluronic corrosive (HA), is a characteristic glycosaminoglycan orchestrated by type B synovial cells, chondrocytes, and fibroblasts and emitted into the synovial liquid. It gives thick grease, has stunning engrossing properties and also, conceivable calming and against oxidant capacities have been depicted.

REGENERATIVE MEDICATION

IA infusions of autologous adapted serum (ACS), platelet-rich plasma (PRP), and mesenchymal foundational microorganisms (MSC) have been tested. Their components of activity is a decrease of fiery responses interceded by cytokines, and the acceptance of anabolism and chondrocyte separation by means of development factors and undeveloped cells contained in it. These strategies are promising and a few investigations have announced them to be protected, very much endured and better than IA fake treatment and HA as far as help with discomfort and knee function^[33,34,35,36].

SURGERY

Surgery is utilized where clinical treatment has arrived at its limits. Surgical signs and decision of treatment depend on side effects (e.g., agony and knee work), OA stage, and patient-related factors, for example, age, level of physical movement, and patient's comorbidities. Radiological proof of OA alone doesn't legitimize careful mediation which is shown uniquely in blend with important side effects. At last, it is the patient's level of anguish, in relationship to radiological proof of OA, which decides the time purpose of medical procedure.

ARTHROSCOPIC LAVAGE AND DEBRIDEMENT

Arthroscopic strategies incorporate lavage and debridement of the knee (e.g., shaving of unpleasant ligament or smoothening of the declined meniscus)^[37,38].

LIGAMENT REPAIR TECHNIQUES

Damaged articular ligament has just constrained or no recuperating limit. Fix of the ligament surface has along these lines been proposed [39].

BONE MARROW STIMULATING TECHNIQUES

The entrance of the sub-chondral lamina has been appeared to advance ligament fix tissue; undoubtedly, pluripotent undeveloped cells emerging from the subchondral bone marrow may advance chondrogenesis in the deformity zone. This method improves chondralre-emerging and exploits the mending capability of the body^[40].

OSTEOCHONDRAL TRANSPLANTATION TECHNIQUES

Remaking of a cartilaginous surface or of osteocartilaginous deformities should be possible by transplantation of osteochondral joins. The join can be autologous or allogeneic^[41].

AUTOLOGOUS CHONDROCYTE IMPLANTATION (ACI)

Autologous chondrocytes are re-embedded underneath a periosteal fold. Fundamental signs for ligament fix procedures are restricted size ligament injuries particularly in more youthful patients^[42].

OSTEOTOMIES AROUND THE KNEE

Osteotomies around the knee are an acknowledged technique for the treatment of unicompartmental OA with related varus or valgus deformity^[43]. Osteotomy turned into a standard treatment choice for unicompartmental OA of the knee^[44].

UNICOMPARTMENTAL KNEE ARTHROPLASTY (UKA)

UKA is shown in situations where OA includes just one of the three compartments of the knee: the average tibiofemoral, horizontal tibiofemoral or patellofemoral compartment [45]. The commonest UKA replaces the contact surfaces of the average tibiofemoral compartment with two metallic prosthetic gadgets and supplements a polyethylene trim between them [46]. As of recent times, it has been the principal line technique for end-stage knee OA [47].

MINIMAL INVASIVE SURGERY (MIS)

Most knee arthroplasties are embedded through a parapatellar average arthrotomy with parting of the quadriceps ligament and the retinaculum/case adjacent to the patella and patellar ligament. The patella is generally everted. The supposed "smaller than expected intrusive medical procedure" abstains from the parting of the quadriceps ligament.

EMBED FIXATION

Established obsession of complete knee supplanting is a standard technique with great long haul toughness.

Because of the advancement of careful methods and improved embed innovation, the result and capacity of TKA have improved. For effective results, great arrangement of the tibial and

femoral parts is fundamental, prompting lower wear of the prosthesis. TKA has become a fruitful treatment for cutting edge and suggestive knee OA, especially in old patients.

Joint substitution is the last answer for some individuals, giving torment-free and working joints for up to 20 years^[48,49,50,51].

CONCLUSION:

Osteoarthritis (OA) is a major public health concern around the world and a devastating condition that leads to pain, diminished quality of life and high health care costs among older adults. Advances in the understanding of osteoarthritis have revealed new aspects of the pathogenesis and progression of the disease. Various risk factors found are useful in identifying patients with the greatest risk of developing OA and patients with a high risk of disease progression.

REFERENCES:

- 1. Peach CA, Carr AJ, Loughlin. Recent advances in the genetic investigations of Osteoarthritis. Trends in Molecular Medicine 2005;11:186-91.
- 2. Kloppenburg M, Berenbaum F. Osteoarthritis year in review 2019: epidemiology and therapy. Osteoarthritis and Cartilage2020;28(3): 242-248.
- 3. Johanne Martel-Pelletier. Pathophysiology of osteoarthritis. Osteoarthritis and Cartilage 2004; 12:S31–S33.
- 4. Helmick CG, Felson DT, Lawrence RC, *et al.* Estimates of the prevalence of arthritis and other rheumatic conditions in the United States, Part I. Arthritis and Rheumatology 2008; 58(1): 15–25.
- 5. Lawrence RC, Felson DT, Helmick CG, *et al.* Estimates of the prevalence of arthritis and other rheumatic conditions in the United States, Part II. Arthritis and Rheumatology2008; 58(1): 26–35.
- 6. Van Saase JL, van Romunde LK, Cats A. Epidemiology of osteoarthritis: Zoetermeer survey. Comparison of radiological osteoarthritis during a Dutch population thereupon in 10 other populations. Annals of the Rheumatic Diseases1989;48(4):271–80.
- 7. Oliveria SA, Felson DT, Reed JI. Incidence of symptomatic hand, hip, and knee osteoarthritis among patients during a health maintenance organization. Arthritis and Rheumatology1995;38(8):1134–41.
- 8. Anna Litwic, Mark H Edwards, Elaine M Dennison, Cyrus Cooper. Epidemiology and burden of osteoarthritis. British Medical Bulletin2013;105:185-99.
- 9. Srikanth VK, Fryer JL, Zhai G. A Meta-analysis of sex differences in prevalence, incidence and severity of osteoarthritis. Osteoarthritis Cartilage 2005;13(9):769–81.
- 10. Cauley J, Kwoh C, Egeland G. Serum sex hormones and severity of osteoarthritis of the hand. The Journal of Rheumatology 1993; 20(7): 1165–1170.
- 11. Sowers MF, Hochberg M, Crabbe JP. Association of bone mineral density and sex hormone levels with osteoarthritis of the hand and knee in premenopausal women. American Journal of Epidemiology 1996; 143(1): 38–47.
- 12. Sowers M, Lachance L. The roles of vitamins A, C, D, and E. Rheumatic Diseases Clinics of North America 1999; 25(2): 315–332.
- 13.McAlindon TE, Jacques P, Zhang Y. Do antioxidant micronutrients protect against the development and progression of knee osteoarthritis?. Arthritis and Rheumatism 1996; 39(4): 648–656.
- 14. Nigel Arden, Michael C. Nevitt. Osteoarthritis: Epidemiology. Best Practice & Research Clinical Rheumatology, 2006;20(1):3-25.

- 15. Cooper C, Snow S, McAlindon TE. Risk factors for the incidence and progression of radiographic knee osteoarthritis, Arthritis and Rheumatism 2000; 43(5): 995–1000.
- 16. Buckwalter JA, Lane LE. Athletics and osteoarthritis. The American Journal of Sports Medicine 1997;25(6):873–81.
- 17. Kellgren J, Lawrence J. Atlas of ordinary Radiographs. The Epidemiology of Chronic Rheumatism, Vol. 2. Oxford: Blackwell Scientific Publications. 1963.
- 18. DeGroot J, Bank, RA, Tchetverikov I. Molecular markers for osteoarthritis: the road ahead. Current Opinion in Rheumatology 2002;14(5):585–9.
- 19. Vignon E, Garnero P, Delmas P. Recommendations for the registration of medicine utilized in the treatment of osteoarthritis: an update on biochemical markers. Osteoarthritis Cartilage 2001;9:289–93.
- 20. Johanne Martel-Pelletie. Pathophysiology of osteoarthritis. OsteoArthritis and Cartilage 2004; 12: 31–33.
- 21. Sokoloff L (eds). The joints and synovial fluid, New York: Academic Press1980,177-238.
- 22. Garstang SV, Stitik TP. Osteoarthritis Epidemiology, Risk Factors, and Pathophysiology. American Journal of Physical Medicine &Rehabilitation 2006;85(11):2-11.
- 23. Brandt KD, Dieppe P, Radin E. Etiopathogenesis of osteoarthritis, Medical clinics of North America 2009; 93(1):1-24.
- 24. Yuan GH, Tanaka M, Masuko-Hongo K, Shibakawa A, Kato T, Nishioka K, Nakamura H, *et al.* Characterization of cells from pannus-like tissue over articular cartilage of advanced osteoarthritis. Osteoarthritis Cartilage 2004; 12(1):38-45.
- 25. Sellam J, Berenbaum F. The role of synovitis in pathophysiology and clinical symptoms of osteoarthritis. Nature Reviews Rheumatology 2010: 6(11):625-635.
- 26. Hasegawa M, Segawa T, Maeda M, Yoshida T, Sudo A. Thrombin-cleaved osteopontin levels in synovial fluid correlate with disease severity of knee osteoarthritis. The Journal of Rheumatology 2011; 38(1): 129-134.
- 27. Englund M, Roemer FW, Hayashi D, Crema MD, Guermazi A. Meniscus pathology, osteoarthritis and the treatment controversy, Nature Review Rheumatology 2012; 8(7):412-419.
- 28. Scott DL, Shipley M, Dawson A, Edwards S, Symmons DP, Woolf AD. The clinical management of atrophic arthritis and osteoarthritis: strategies for improving clinical effectiveness. The British Journal of Rheumatology 1998;37(5):546-554.
- 29. Thomas KS, Muir KR, Doherty M, Jones AC, O'Reilly SC, Bassey EJ. Home based exercise programme for knee pain and knee osteoarthritis: randomised controlled trial. British medical journal2002;325(7367):752.
- 30. Esser S, Bailey A. Effects of exercise and physical activity on knee osteoarthritis. Current Pain and Headache Reports 2011;15(6):423-30.
- 31. Jevsevar DS. Treatment of osteoarthritis of the knee: evidence-based guideline, 2nd edition. The Journal of the American Academy of Orthopaedic Surgeons 2013;21(9):571–576.
- 32. Juan C Mora, Rene Przkora, Yenisel Cruz-Almeida. Knee osteoarthritis: pathophysiology and current treatment modalities. Journal of Pain Research 2018; 11: 2189–2196.
- 33.Leslie J Crofford. Use of NSAIDs in treating patients with arthritis. Arthritis Research & Therapy 2013; 15(3): S2.
- 34. Dimitrios Filippiadis, George Charalampopoulos, Argyro Mazioti, Efthymia Alexopoulou, Thomas Vrachliotis, Elias Brountzos, Nikolaos Kelekis, Alexis Kelekis, *et al.* Interventional radiology techniques for pain reduction and mobility improvement in patients with knee osteoarthritis. Diagnostic and Interventional Imaging 2019;100(7-8):391-400.
- 35. Ayhan E, Kesmezacar H, Akgun I. Intraarticular injections (corticosteroid, mucopolysaccharide, platelet rich plasma) for the knee osteoarthritis. World Journal of Orthopedics 2014;5(3):351–361.
- 36. Richards MM, Maxwell JS, Weng L, Angelos MG, Golzarian J. Intraarticular treatment of knee osteoarthritis: from anti-inflammatories to products of regenerative medicine. The Physician and Sports medicine 2016;44(2):101–108.
- 37. Chang RW, Falconer J, Stulberg SD, Arnold WJ, Manheim LM, and Dyer AR. A randomized, controlled trial of arthroscopic surgery versus closed- needle joint lavage for patients with osteoarthritis of the knee. Arthritis and Rheumatism1993;36(3):289-296.
- 38. Ogilvie-Harris, Fitsialos DP. Arthroscopic management of the degenerative knee. Arthroscopy 1991;7(2):151-157.

- 39. Widuchowski W, Lukasik P, Kwiatkowski G. Isolated full thickness chondral injuries. Prevalance and outcome of treatment. A retrospective study of 5233 knee arthroscopies. Acta Chirurgiae Orthopaedicae et Traumatologiae Cechoslovaca2008;75(5):382-386.
- 40. Pridie KH. A method of resurfacing osteoarthritic knee joints. Journal of Bone and Joint Surgery 1959;41:618-619.
- 41. Hangody L, Feczk P, Bartha L, G. Bod, and Kish G. Mosaicplasty for the treatment of articular defects of the knee and ankle. Clinical Orthopaedics and Related Research 2001;391:S328-S336.
- 42. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. The New England Journal of Medicine 1994;331(14):889-895.
- 43. Brinkman JM, Lobenhoffer P, Agneskirchner JD, Staubli AE, Wymenga AB, Van Heerwaarden RJ. Osteotomies around the knee Patient selection, stability of fixation and bone healing in high tibial osteotomies. The Journal of Bone and Joint Surgery. British 2008;90(12):1548-1557.
- 44. Coventry MB. Osteotomy of the upper portion of the tibia for degenerative arthritis of the knee. A preliminary report. The Journal of Bone and Joint Surgery 1965;47:984–990.
- 45. Marmor L. Marmor modular knee in unicompartmental disease. Minimum four-year follow-up. Journal of Bone and Joint Surgery 1979;61(3):347–353.
- 46. Murray DW. Unicompartmental knee replacement: now or never? Orthopedics2000;23(9): 979–980.
- 47. Keating EM, Meding JB, Faris PM, Ritter MA. Long-term follow up of nonmodular total knee replacements. Clinical Orthopaedics and Related Research 2002;404:34–39.
- 48. Lonner JH,Lotke PA. Aseptic complications after total knee arthroplasty. The Journal of the American Academy of Orthopaedic Surgeons 1999;7(5): 311–324.
- 49. Gandhi R, Tsvetkov D, Davey JR, Mahomed NN. Survival and clinical function of cemented and uncemented prostheses in total knee replacement: a meta-analysis. Journal of Bone and Joint Surgery 2009;91(7): 889–895.
- 50. Bauwens K, Matthes G, Wich M. Navigated total knee replacement: a meta-analysis. Journal of Bone and Joint Surgery 2007;89(2):261–269.
- 51. Collier MB, Engh CA, McAuley JP, Engh GA. Factors associated with the loss of thickness of polyethylene tibial bearings after knee arthroplasty. Journal of Bone and Joint Surgery 2007;89(6):1306–1314.