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Osteoarthritis and its treatment modalities: A review of literature

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Abstract

Osteoarthritis (OA) is a common chronic condition resulting in pain, fatigue, functional limitations, increased healthcare utilization and high economic costs to society. The burden of OA is projected to increase due in part to obesity and ageing population. While the prevalence of OA increases with age. Pathologically, knee OA is characterized by structural changes in and around the knee joint. The predominant structural changes are the loss of cartilage and the formation of osteophytes. These changes are easily demonstrated radiologically, and objective measures of disease severity are based on the amount of joint space loss (a reflection of cartilage loss) and the presence of osteophytes. For the management of OA, surgical or conservative method is used which is primarily directed toward pain control and reduction in functional limitation.

Keywords: Osteoarthritis (OA), treatment modalities, pain, fatigue, functional limitations

Introduction

Osteoarthritis (OA) is a prevalent chronic disease that causes symptoms such as pain, fatigue, functional restrictions, increased healthcare consumption, and significant societal expenses. Because of population ageing and obesity, the burden of OA is expected to rise, while the frequency of OA rises with age^[1].

Patients typically report of pain that is aggravated by physical activity and released when the patient takes rests; but, in progressive form, pain can also exist while the patient is passive. When morning stiffness occurs, it usually lasts little longer than half an hour. Symptom severity might range from an asymptomatic ailment identified radiographically to severe pain with functional limitations. It is possible to arrive at a sensible conclusion based on the patient's anamnesis and examination diagnosis^[2].

Repetitive mechanical stresses and ageing are linked to the etiology of OA. There are three primary sub-groups of etiological factors: age: Most common in those aged 55 to 64, sex: - Females have a higher prevalence; body mass: Obese people have a higher prevalence than non-obese people. Other variables that contribute to the development of medial OA include hereditary and indigenous unfavorable mechanical reasons such as joint trauma, professional and recreational misuse, alignment, and post-meniscectomy and topographical factors^[1, 3]. Besides Genetic variables are closely linked to the development of OA; differences in genetic structure lead to chondrocyte alterations, which eventually contribute to osteoarthritis^[4, 5].

Tenderness on palpation, stiffness, crepitus with movement, joint discomfort, bone enlargement, aberrant orientation, restricted range of joint mobility, and joint effusion are some of the clinical signs^[1, 2]. These symptoms make it difficult for the person to walk, squat, or climb stairs. Because of arthritis of uneven joint surfaces, crepitation can be detected during movement. According to recent US data, half of patients with symptomatic knee OA present by the age of 55^[1, 5].

The pathogenic element in the process of cartilage degeneration has been considered to be subchondral bone sclerosis in the early stages of OA, which may involve micro fractures^[6]. Structure anomalies in and nearby the knee joint describe the pathophysiology of knee OA. The growth of osteophytes and the loss of cartilage are the two most significant structural alterations.

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These changes are plainly visible on radiographs, and the presence of osteophytes and the amount of joint space loss are used to determine the severity of the condition (a reflection of cartilage loss) [7-9].

Patient history, physical examination, laboratory, and radiologic results are all used to diagnose knee OA. However, most patients may be diagnosed with knee OA using only normal Röntgen rays. Computed tomography, ultrasonography, magnetic resonance imaging, and a bone scan are examples of imaging modalities that can provide alternate or additional information [10].

The plain radiograph is the most important test for diagnosing knee OA and determining the severity of the condition. Radiography has the following advantages: it is relatively safe and cost-effective, and it is widely available [11]. Osteophytes, subchondral bone sclerosis, limited joint space, osteochondral bodies, cyst formation, and bone deformities are all classic radiographic characteristics of knee OA. The Kellgren and Lawrence system is a five-grade system for grading the severity of knee osteoarthritis (OA). Kellgren *et al.* proposed the categorization in 1957, and WHO recognized it in 1961. The characteristic of OA is osteophytes, which grow at the joint borders as a result of endochondral ossification. They can be interpreted as a repair attempt, implying that aberrant joint loading has been redistributed [12].

On entire limb standing antero-posterior (AP) radiographs, a mechanical tibiofemoral axis narrower than 180° is one of the distinguishing markers of knee varus distortions. One of the most frequent indications in people with knee OA is a narrowing of the medial joint space. These symptoms are observed in 74% of people with idiopathic OA [13]. Even in healthy knees, the medial compartment is said to withstand 60 to 80 percent of the joint's load [14]. Though, no precise material exists that explains what causes the joint's unequal stress distribution. The joint's load is distributed along the mechanical axis, which is located medial to the knee's center.

The treatment of osteoarthritis (OA) can be conservative or surgical, and the goal is to reduce functional limitations and pain. The patient's education is incorporated into conservative therapy. The doctor should talk about the disease process, as well as the risks and benefits of various therapy alternatives. Educating the patient and equipping them with skills that will help them make beneficial lifestyle choices. Weight loss, achieved through exercise, physiotherapy, and proper food habits, improves pain and function [15]. Assistive equipment such as a cane, frame, or wheeled walker are used. For pain alleviation, transcutaneous electrical nerve stimulation (TENS) is recommended [16].

Oral administration of paracetamol, which may cause upper gastrointestinal side effects and a modest decrease of renal function, is one pharmacological method for symptomatic therapy. Antidepressants and Tricyclic compounds are routinely recommended as chronic pain in OA is typically accompanied by anxiety, insomnia, and depression. If paracetamol or NSAIDs are not successful, opioids such as tramadol or codeine might be tried. Patients with acute flares and knee effusions may benefit from the use of long-acting intra-articular (IA) corticosteroids. IA steroids provide immediate pain relief and can persist for four to twelve weeks [16, 17].

Patients with limited function and persistent pain who have failed to respond to non-surgical treatments may benefit from joint replacement surgery, which has a significant impact on their quality of life [17]. Although many patients who had total knee arthroplasty (TKA) reported increased function and fewer

symptoms, many nevertheless report some level of persistent discomfort. TKA is both costly and complicated, thus it may necessitate a second modification [18].

In young patients with OA of medial compartment of knee, HTO was surgical therapy of choice, with goal of reestablishing alignment and postponing the need for TKA [18]. HTO has a number of drawbacks, including a longer time for complete weight bearing, the likelihood non-union or obstructed union, peroneal nerve palsy, and wound infection [18].

Although arthroscopic debridement decreases pain, it is linked to a high likelihood of recurrence [19]. Arthroplasty is most effective in individuals over the age of 60, however it can be dangerous [20]. Correcting a varus-valgus deformity with osteotomy is advantageous, normalizing the alignment of the lower limbs, lowering the amount of tension on the articular surfaces, reducing joint discomfort, delaying the onset of joint degeneration, as well as postponing and preventing joint replacement; In order to avoid major surgery, many younger patients with early knee OA are given a variety of nonsurgical treatments, such as exercise and physical therapy, bracing, or those, non-steroidal anti-inflammatory drugs (NSAIDs), and intraarticular supplements or corticosteroid injection. The goals of these therapy options are to relieve pain and improve overall function. Some of these modalities may have a disease-modifying effect by altering the mechanical environment of the knee. For optimal use of certain nonsurgical modalities, knowledge of evidence-based practice standards, careful patient selection, patient education, and adequate long-term follow-up are all essential [21].

Due to the poor early results of unicompartmental knee arthroplasty (UKA) in the 1970s and early 1980s, many surgeons treated unicompartmental OA with either HTO or TKA. UKA has experienced a renaissance. In last 10 to 15 years, the indicators for UKA have shifted dramatically. Low-demand patients over the age of 60 with a low BMI, intact ligamentous structures, and isolated medial compartment OA but no flexion or extension contractures are traditionally candidates for UKA. The indications have widened to include younger patients, patients with mild patellofemoral OA, and some patients with anterior cruciate ligament (ACL) deficiency, thanks to advancements in UKA prosthetic design, polyethylene wear characteristics, and surgical technique. Recent research has focused on the outcomes of UKA in a younger patient population. Schai *et al.* looked at 28 UKAs in patients aged 60 and up, with an average age of 52 at the time of surgery. Two to six years of follow-up was required. Ninety percent of patients had good or excellent results in terms of function and pain reduction. According to the Tegner and Lysholm score, average activity level improved somewhat from 2.3 points preoperatively to 2.7 points at followup. 9 (32%) of the 28 knees had radiolucent lines around the tibial component, and 2 had partial radiolucent lines on the femoral side at the bone-cement contact. There was no link between activity level and the presence of tibial radiolucent lines, according to the researchers. Two patients required revision due to loosening of the femoral component at the prosthesis-cement contact; one got another UKA and the other was converted to TKA. One tibial component was asymptomatic, slowly increasing radiolucency. The authors concluded that UKA was successful in middle-aged patients, but that it was not superior to TKA in terms of survival. Pennington *et al* looked at 41 patients over the age of 60 who had had UKA (average age, 54 years). All of the patients who had the treatment were regarded as physically active and involved in high-demand activities. The Hospital for Special

Surgery score was outstanding in 93% of patients after an 11-year follow-up and good in 7%. Three patients needed revision surgery: two for severe tibial osteolysis and one for persistent knee discomfort and a progressing tibial radiolucent line. Nine knees demonstrated OA development in the un-resurfaced compartment, but none of them required conversion to TKA, and none of the patients' Hospital for Special Surgery scores worsened. At 11 years, the total survival rate was 92 percent. Chronic ACL deficit causes medial meniscal tears and medial compartment OA, with few therapeutic options available. Because eccentric loading and severe wear occurred on the tibial polyethylene component, ACL insufficiency was traditionally a contraindication to UKA for the therapy of medial compartment OA. However, the literature does not always support this. Hernigou and Deschamps looked back on the results of 99 UKAs after a 16-year follow-up period. The ACL was judged normal in 50 knees at the time of the arthroplasty, injured in 31, and missing in 18. At the final follow-up, 11 of the 18 knees with ACL deficit at the time of surgery had not failed. In these 11 knees, the average posterior tibial slope was 5°. Seven knees were reconstructed after the ACL was removed during the arthroplasty procedure. The tibial prosthesis was implanted with a posterior slope of >8° in these knees. When the tibial component had a slope of 7°, the authors found that the outcomes in the ACL-intact and ACL-deficient groups were equivalent. Reconstructing the ACL in conjunction with the UKA is another option for people with ACL deficit. Pandit *et al* compared the results of 15 patients who had ACL reconstruction with UKA to a group of 15 individuals who had an undamaged ACL. There was no difference between the groups in terms of Oxford Knee Scores and Knee Society scores 2.5 years following the procedure. The functional Knee Society score for both groups was 96. (Out of 100). Despite the fact that the study was very short-term, no patient in either group had radiologic signs of loosening. *In vivo* kinematics of knees that had ACL repair and UKA were also shown to be equal in a step-up exercise and in deep knee flexion. Because the early findings are promising, combined therapy of ACL deficit with medial compartment OA with ACL repair and UKA demands additional exploration and long-term follow-up investigations^[21].

HTO combines valgus proximal tibial osteotomy with supracondylar femoral osteotomy, with a satisfaction rate of up to 60% after ten years^[22, 23].

Because of the lateral support supplied by the fibula-soft tissue combination to the osteoporotic tibia^[24, 25], bilateral tibial plateau deterioration and non-uniform settling are possible, as an outcome, the load transfers from the usual sharing to medial side of the medial plateau, causing varus defect in knee and ultimately exacerbating and progressing the medial compartment KOA. Keeping this in mind, proximal fibular osteotomy may be beneficial in reducing the loading strength on the medial compartment, resulting in improved knee joint care for medial compartment OA.

The proximal fibular osteotomy procedure, which begins and encourages the advancement of Knee Osteoarthritis, is based on the 'theory of differential settlement' or 'non uniform.

A novel surgical procedure According to Zhang *et al.* in 2015, proximal fibular osteotomy (PFO) is thought to reduce pain and enhance joint function in knee OA patients although proximal fibular osteotomy is a straightforward technique, it must be done with caution to avoid peroneal nerve injury.

Regenerative aspects

Traditional OA treatment (*e.g.*, arthroscopic debridement, micro

fracture, autologous or allogeneic cartilage transplantation, chondrocyte transplantation) is primarily symptomatic treatment and pain management, which cannot contribute to regenerating degenerated cartilage or reducing joint inflammation. These days' Mesenchymal stem cells (MSCs) have turned into the most extensively explored new therapeutic drugs in cell-based OA treatment as a result of their ability to differentiate into chondrocytes and their immunomodulatory properties. Mesenchymal stem cells (MSCs) have the potential of self-renewal and directional differentiation, which can repair cartilage tissue and suppress chondrocyte secretion of inflammatory factors and homing characteristics, which make MSCs the ideal seed cells for gradual OA treatment. Platelet-rich plasma (PRP) with the active substance can promote cell proliferation, collagen synthesis and inflammatory chemotaxis. Thus, it is conducive to tissue repair and can assist tissue reconstruction. Pre-clinical studies have verified that PRP/MSCs can also improve knee joint function, and the repaired tissue exhibits good compatibility with the original articular facial cartilage tissue by MRI analysis.

Conclusion

One of the most frequent joint illnesses is osteoarthritis of the knee, and it causes a lot of pain and immobility. High tibial osteotomy and unicompartmental knee replacement are two of the few surgical managements for treating medial compartment arthritis of the knee. The varus deformity associated with medial compartment arthritis of knee can be managed with a high tibial osteotomy, but it comes with a lengthier recovery time and a period of non-weight bearing walking till the site of osteotomy gets healed. Both recurrence of varus deformity and the requirement for a total knee arthroplasty revision due to persistent pain are likelihoods. Detailed clinical and radiological assessment, careful preoperative planning, meticulous dissection, precision in surgical technique are critical in achieving good results and minimizing the complication.

References

1. Losina E, Weinstein AM, Reichmann WM, Burbine SA, Solomon DH, Daigle ME, *et al.* Lifetime risk and age at diagnosis of symptomatic knee osteoarthritis in the US. *Arthritis Care Res (Hoboken)*. 2013;65(5):703-11.
2. Ashman RF, Ferguson PJ, IJdo JW, Lawry GV II. *Rheumatic Diseases: Diagnosis and Management*; Weinstein, Stuart L; Buckwalter, Joseph A; Turek's Orthopaedics: Principles and Their Application, 6th Edition: Lippincott Williams & Wilkins, 2005, 154-55.
3. Spector TD, Cicuttini F, Baker J, Loughlin J, Hart D. Genetic influences on osteoarthritis in women: a twin study. *British Medical Journal*, 1996;312(7036):940-44.
4. Kraan PM, Davidson ENB, Blom A, Berg WB. TGF-beta signaling in chondrocyte terminal differentiation and osteoarthritis: modulation and integration of signaling pathways through receptor-Smads. *Osteoarthritis Cartilage*. 2009;17(12):1539-45.
5. Valdes AM, Spector TD, Tamm A. Genetic variation in the SMAD3 gene is associated with hip and knee osteoarthritis. *Adv Rheumatol*. 2010;62(8):2347-52.
6. Burr DB, Radin EL. Microfractures and microcracks in subchondral bone: are they relevant to osteoarthritis? *Rheum Dis Clin North Am*. 2003;29:675-85.
7. Goldring MB, Goldring SR. Osteoarthritis. *J Cell Physiol*. 2007;213:626-64.
8. Arokoski JP, Jurvelin JS, Väättäinen U, Helminen HJ.

- Normal and pathological adaptations of articular cartilage to joint loading. *Scand J Med Sci Sports*. 2000;10:186-98.
9. Felson DT. An update on the pathogenesis and epidemiology of osteoarthritis. *Radiol Clin North Am*. 2004;42:1-9.
 10. Iagnocco G, Meenagh L, Riente, *et al*. Ultrasound imaging for the rheumatologist XXIX. Sonographic assessment of the knee in patients with osteoarthritis, *Clinical and Experimental Rheumatology*. 2010;28(5):643-46.
 11. Loeuille D, Chary-Valckenaere I, Champigneulle J, Rat AC, Toussaint F, Pinzano-Watrin A, *et al*. Macroscopic and microscopic features of synovial membrane inflammation in the osteoarthritic knee: correlating magnetic resonance imaging findings with disease severity. *Arthritis Rheum*. 2005;52:3492-501.
 12. Kellgren JH, Lawrence JS. Radiological Assessment of Osteo-Arthrosis. *Annals of the Rheumatic Diseases*. 1957;16(4):494-502.
 13. Shiozaki H, Koga Y, Omori G, Yamamoto G, Takahashi HE. Epidemiology of osteoarthritis of the knee in a rural Japanese population. *Knee*. 1999;6(3):183-88.
 14. Ahlbäck S. Osteoarthrosis of the knee: a radiographic investigation. *Acta Radiol*. 1968;277:7-72.
 15. Spector TD, Hart DJ, Doyle DV. Incidence and progression of osteoarthritis in women with unilateral knee disease in the general population: the effect of obesity. *Ann Rheum Dis*. 1994;53(9):565-8.
 16. W-Dahl A, Robertsson O, Lidgren V. Surgery for knee osteoarthritis in younger patients. *Acta Orthop*. 2010;81:161-4.
 17. Roos EM, Juhl CB. Osteoarthritis 2012 year in review: rehabilitation and outcomes. *Osteoarthr Cartil*. 2012;20:1477-83.
 18. Prieto-Alhambra D, Javaid MK, Judge A. Hormone replacement therapy and mid-term implant survival following knee or hip arthroplasty for osteoarthritis: a population-based cohort study. *Ann Rheum Dis*. 2015;74:557-63.
 19. Feeley BT, Gallo RA, Sherman S, Williams RJ. Management of osteoarthritis of the knee in the active patient. *J Am Acad Orthop Surg*. 2010;18:406-16.
 20. Bae DK, Song SJ, Kim KI, Hur D, Jeong HY. Mid-term survival analysis of closed wedge high tibial osteotomy: A comparative study of computer-assisted and conventional techniques. *Knee*. 2016;23:283-88.
 21. Amendola A, Bonasia DE. Results of high tibial osteotomy: review of the literature. *Int Orthop*. 2010;34:55-60.
 22. Zhang Y, Li C, Li J, *et al*. The pathogenesis research of non-uniform settlement of the tibial plateau in knee degeneration and varus. *J Hebei Med Univ*. 2014;35(2):218-19
 23. Zheng Z, Sun Y, Zhang X, Chen W, Li S, Zhang Y. The pathogenesis and clinical imageology research of the knee osteoarthritis. *J Hebei Med Univ*. 2014;35(5):599-600.
 24. Yang ZY, Chen W, Li CX, Wang J, Shao DC. Medial compartment decompression by fibular osteotomy to treat medial compartment knee osteoarthritis: a pilot study. *Orthopedics*. 2015;38:1110-14.
 25. Tong G, Xie Q. Clinical observations of medial compartment knee osteoarthritis by proximal fibular osteotomy with arthroscopy. *Zhonghua Yi Xue Za Zhi*. 2016;96(43):3508-10.