https://doi.org/10.52889/1684-9280-2024-1-71-21-29 UDC 616-089.23; 616-001; 615.477.2 IRSTI 76.29.41

A Descriptive Overview

Diagnosis and Treatment of Knee Osteoarthritis: Modern Aspects

Aida Baibusunova¹, Gaukhar Nyssanbay², Dmitriy Shwartz³, Meruert Zhakeeva⁴

¹ Student of the School of Medicine, Astana Medical University, Astana, Kazakhstan. E-mail: baibusunova@ca-amu.kz
² Student of the School of Medicine, Astana Medical University, Astana, Kazakhstan. E-mail: n.n.gauhar@gmail.com
³ Assistant of the Department of Traumatology and Orthopedics of Astana Medical University, Astana, Kazakhstan. E-mail: shwartz_dima81@mail.ru

⁴ Assistant of the Department of Internal Diseases with courses in nephrology, hematology, allergology and immunology, Astana Medical University, Astana, Kazakhstan. E-mail: mikena90@mail.ru

Abstract

Osteoarthritis of the knee joint (KOA) is a common chronic joint disease that most often affects adults over the age of 60. Recent studies indicate its progressive nature, involving structural changes in various components of the joint, including articular cartilage, synovial membrane and ligaments.

Globally, about 250 million people suffer from KOA, with the incidence of KOA increasing due to asymptomatic cases, obesity and other factors. Women are more likely to be affected by this disease than men. OA is becoming one of the main causes of disability in the world, accounting for 85% of the burden of KOA. Approximately 10% of people over the age of 60 suffer from KOA, which leads to movement restrictions

The purpose of the review is to highlight recent studies on imaging and current treatments for KOA, as well as an analysis of the prevalence of this disease.

Due to the severity of the process and the pain syndrome, non-surgical methods are often insufficient. Joint replacement is an important method of treatment at the terminal stage. However, other methods, such as corticosteroid injections, hyaluronic acid and the use of cells, can slow the progression of the disease. Timely diagnosis plays a key role, given that KOA can be asymptomatic in the early stages. It is important to take into account the symptoms, clinical data and results of various imaging techniques for effective disease management.

Keywords: osteoarthritis of the knee joint, diagnosis of osteoarthritis, treatment of osteoarthritis, total endoprosthesis, mesenchymal stem cells.

Corresponding author: Meruert Zhakeeva, Assistant of the Department of Internal Diseases with courses in nephrology, hematology, allergology and immunology, Astana Medical University, Astana, Kazakhstan. Postal code: Z10K8Y7

Address: Kazakhstan, Astana, Beibitshilik st 49A Phone: +7 (777) 997 64 37 E-mail: mikena90@mail.ru

> J Trauma Ortho Kaz 2024; 1 (71): 21-29 Recieved: 12-12-2023 Accepted: 14-02-2024



This work is licensed under a Creative Commons Attribution 4.0 International License

Introduction

Knee osteoarthritis (KOA) is the most common chronic multifactorial joint disease, which mostly affects adults 60 years of age or older [1]. According to the latest research, osteoarthritis (OA) is the progressive musculoskeletal condition, that injures dominant weightbearing joints, like the hips or knees joints [2-4]. This inflammatory disease includes structural changes in the articular cartilage, subchondral bone, Hoffa's adipose tissue, synovia, ligaments and muscles, therefore it is characterized not only as a mechanical degeneration of the articular cartilage, but also as concomitant structural and functional changes in the entire joint, including the meniscus and periarticular ligament [5,6].

Approximately 250 million people in all regions of the world suffer from OA, in that case the incidence of KOA increased significantly over the decade due to the higher prevalence of asymptomatic cases, as well to obesity and other risk factors [7]. Women suffer more often, which is confirmed by data on the prevalence of KOA at the age of 60 years and older, in men it is about 10% and 13% in women [1].

According to the GBD 2015 Disease and Injury Incidence and Prevalence Collaborators, KOA is the main cause of the burden of osteoarthritis worldwide and estimated around 85% [8]. OA is one of the leading causes of increasing the global years lived with disabilities (YLD) [8]. Approximately 10% of the population over the age of 60 years is affected by KOA, and 80% of this population have

Causes and Risk Factors

Although KOA was thought to be a normal consequence of aging and a mechanical consequence of "wear and tear", it has therefore been characterized as degenerative. But now known that the development of KOA is associated with a multifactorial complex interaction of constitutional and mechanical factors, including joint integrity, lifestyle, genetic predisposition, local inflammation, mechanical influences, as well as cellular and biochemical processes [12-14]. The development of KOA is closely related to the aging of the body, since radiological signs occur in more than 75% of people over 75 years of age [15]. Obesity (elevated body mass index (BMI)), joint displacement and instability leading to increased mechanical stress are strong risk factors for KOA [16,17]. The medial tibiofemoral compartment, the lateral tibiofemoral compartment, and the patellofemoral joint work together to form an articulation. However, due to anatomical features, excessive stress on the knee joint can adversely affect the functionality of the knee joint. Therefore, long kneeling, squatting, weight lifting, long-distance running, football and other professional sports are associated with an increased risk of developing KOA and frequent injuries. It has been established that previous traumatization of the knee joints increases the risk of developing KOA by 3.86 times due to defects in cartilage, meniscus, and rupture of the anterior cruciate ligament (ACL) [18].

In turn, the lack of load also increases the prevalence of KOA, because traumatization of the knee joint occurs due to weak joints and less stable ligaments, however, this risk factor is less influential than previous injuries [19]. Thinner

Diagnosis

KOA is usually accompanied by pain and stiffness in the affected joints, mainly in the morning or after prolonged sitting [25]. Pain disappears after half an hour, which is a hallmark of other diseases: inflammatory and movement restrictions, and 25% have functional limitations that compromise the performance of daily activities [9,10].

Due to the fact that KOA is a chronic disease with severe symptoms, such as debilitating pain, non-surgical methods are not enough to improve the quality of life of patients. In case of ineffectiveness of conservative methods of treatment, to improve the quality of life, well-being and physical condition of patients at the terminal stage, joint arthroplasty is used, which is undoubtedly the most important discovery in medicine. However, other methods, such as the administration of corticosteroids, hyaluronic acid, the use of mesenchymal stem cells, as well as plateletrich plasma (PRP) or autologous micro fragmented adipose tissue with a stromal-vascular fraction can slow down the existing condition.

An important aspect in the treatment of KOA is timely diagnosis, due to the fact that KOA in the initial stages can be an asymptomatic disease. Close attention should be paid to the anamnesis and screening for patientreported outcomes such as pain, function and quality of life, clinical findings such as joint tenderness and crepitus, objective measures of physical activity, and various imaging modalities such as magnetic resonance imaging, along with biochemical markers research [11].

The purpose of the study: to highlight recent research regarding KOA on imaging and modern therapies, including prevalence studies.

patellas and differences in the size of the tibial condyles distinguish the kinematics of the knee joint in women, with an increased propensity to develop KOA [19].

KOA is a source of chronic pain, it affects the development of depressive episodes, which are frequent and severe consequences of KOA. Chronic pain creates a vicious circle where the mentally unstable patient restricts physical activity, and physical inactivity contributes to increased knee pain and weight gain [20]. From which it follows that KOA also affects the social burden, increasing it.

Genome-wide association studies have identified 90 genetic risk loci for developing KOA, but these loci are not fully responsible for the development of KOA [21]. The present meta-analysis examined the potential associations between adipokines gene polymorphisms and KOA [22]. The association was observed in gene LEPR 1,137,101. Additionally, limited data revealed that associations may also exist in ADIPOQ rs2241766, VISFATIN rs4730153 and VISFATIN rs16872158 [23]. The relationship between obesity and the prevalence of KOA has been confirmed by a number of studies, in which it was indicated that obesity contributes to an increase in the load on the joint, the development of an inflammatory response, negative consequences in the body and lifestyle change in favor of reducing physical activity [16]. In the near future, according to new studies, the prevalence of obesity will not decrease, which would be associated with an increased incidence of KOA and, accordingly, an increase in the number of surgical interventions [17].

infectious arthritis, bursitis, tendonitis and meniscus rupture. Patellofemoral KOA, which may occur alone or in the presence of tibiofemoral KOA, is characterized by more severe pain when going up and down stairs [26]. KOA effusions are usually absent or mild, while them are more common in inflammatory or infective arthritis and may be accompanied by popliteal or Baker's cyst formation. Bursitis is mostly identified by local pain. Infectious and other inflammatory arthritis can be clearly distinguished from KOA, since in these diseases the number of leukocytes in the synovial fluid exceeds 2000 cells/cm3.

Clinical signs and symptoms of KOA may occur before the appearance of osteophytes and narrowing of the joint space on radiographs, so the absence of radiological signs is not always a reason to exclude KOA from the differential diagnosis. Despite normal radiographs, when the clinical picture is indicative of KOA, the clinician should initiate appropriate treatment [27].

Traditionally, the Kallgren-Lawrence (KL) radiographic classification system is used in the diagnosis of KOA, assessing the degree of development of KOA from level 0 to 4. Consistently, joint space narrowing, sclerosis, cysts, deformities appear, while the appearance of osteophytes indicates the presence of ≥ 2 degree [27].

The WOMAC scale is a questionnaire for selfcompletion by patients, consists of 24 questions

Treatment

Nonsurgical treatment. Conservative treatment is prescribed for all patients diagnosed with KOA, especially those patients who first visit a doctor with knee pain and signs of KOA. Typically, such treatment includes a series of manipulations aimed at reducing pain and increasing joint mobility: reducing daily stress on the affected joint and weight loss, therapeutic exercise, thermal procedures, drug treatment (non-steroidal anti-inflammatory drugs (NSAIDs)), injections of corticosteroids, hyaluronic acid, glucosamine, platelet-rich plasma.

Intra-articular injections. The obvious advantages of intra-articular injections are the low risk of systemic adverse reactions, providing a more direct effect in the form of potential pain reduction and improvement in physical function. This is supported by studies that have shown that such therapy is more effective than oral NSAIDs and other systemic pharmacological treatments [30]. The introduction of corticosteroids has a pronounced immunosuppressive and anti-inflammatory effect, reducing the production of interleukin-1, prostaglandins, leukotrienes, MMP9 and MMP-11 [31-33].

However, the study found that administering corticosteroids may be worse than giving physiotherapy for one year [34]. The introduction of extended-release triamcinolone acetonide has shown that this drug has fewer systemic effects than traditional steroid injections [35-38]. It should be noted that too long-term use of corticosteroid hormones can cause thinning of the skin with the appearance of stretch marks and bruises, high blood pressure, elevated blood sugar levels, cataracts, puffiness of the face (moon-shaped face), thinning of arms and legs, poor wound healing, growth retardation in children, leaching of calcium from bones (with the possibility of developing osteoporosis), weight gain and sudden mood swings [39].

Platelet-rich plasma (PRP) is a safe treatment method at least in the short term (up to 12 months) and is more effective than the administration of hyaluronic acid (HA). A randomized clinical trial (RCT) conducted by Raeissadat et al. compared the long-term effects of intraarticular injections of PRP and HA on clinical outcomes of patients suffering from KOA [40]. After 12 months, the WOMAC pain score significantly decreased in both groups. characterizing the severity of pain (5 questions), stiffness (2 questions) and functional ability (17 questions) of patients with KOA [28].

Traditionally, the intensity of knee pain evaluates with a 10 cm visual analogue scale (VAS). According to this scale, there are four groups: mild (< 4 cm), moderate (4–6 cm), severe (6–8 cm) and very severe (>8 cm) pain. To achieve an adequate assessment of pain and physical function, patients were asked not to take pain-relieving drugs within 48 hours before the study [29].

MRI is used less often in the diagnosis of KOA, as it is more often used in scientific research to detect early KOA. MRI is indicated for conditions requiring faster action, such as subchondral insufficiency, fracture, tumor or infection [19]. Ultrasound is much more sensitive for detecting osteophytes, with a sensitivity of more than 85%, but not as accurate for narrowing the joint space [19]. Being a more economically available research method, compared to MRI, it can be used to track the progression of the disease.

The PRP group showed better results than the HA group (P<0.001), especially at stages 1 and 2 according to KL scale [40].

Weight loss. After a five-year follow-up, it was found that weight loss reduces symptoms and slows the progression of the disease [41]. At the same time, biomechanics is significantly improved, it became known that a decrease in weight by 1 kg leads to a fourfold decrease in the forces acting on the knee [42]. It is important to taking foods such as blueberries, raspberries and pomegranate grass, which can reduce arthritis pain [43]. A randomized controlled trial found that walking reduced pain scores by 1.38 points (on a scale of 0 to 10) (p = 0.003) [44]. Exercise reduces the load on the knee joint during movement, relieving pain and improving joint mobility and rotation [45,46]. By the recent study, telehealth-delivered exercise and diet programs improved pain and function in people with knee osteoarthritis and overweight or obesity [47].

Supporting devices. The effectiveness of unloading knee orthoses in KOA is doubtful due to the fact that most studies on this topic are not randomized [48,49]. A cane, shock-absorbing shoes, and inserts alleviate the symptoms of KOA [50,51]. A recent study by a group of patients wearing the biomechanical footwear device with 2 convex adjustable rubber pads screwed to the outsole at the heel and forefoot showed an improvement in the condition of the patients [52].

Massage and manual therapy. A 60-minute Swedish full-body massage once a week for 8 weeks has shown a consistent effect in improving functional status in adults with osteoarthritis of the knee compared to active controls and usual care [53]. Manual therapy has a positive short-term effect on reducing pain in patients with KOA [54].

Non-steroidal anti-inflammatory drugs and pharmacological treatment. European Association of Osteoporosis and Osteoarthritis Economic Areas (ESCEO) stated non-steroidal anti-inflammatory drugs (NSAIDs), glucosamine sulfate (GS) and chondroitin sulfate (CS) as first-line drugs for the treatment of KOA [55]. Taking NSAIDs is associated with side effects, for example, the daily use of drugs of this group four times increases the risk of bleeding from the upper gastrointestinal tract.

According to a meta-analysis of 280 RCTs, vascular side effects are increased by 1/3 with the administration of a cyclooxygenase-II inhibitor [56,57]. Both gels and creams with ibuprofen, which are clinically effective, are more commonly used in pharmacological treatment [58]. Ketoprofen and diclofenac preparations are well tolerated when used topically [59,60]. Topical administration of diclofenac sodium 2% is associated with good tolerability and long-term clinical benefits [61]. The interleukin-1 inhibitor diacerein and the norepinephrine, serotonin reuptake inhibitor duloxetine (SMD 0.39) can delay the course of the disease, reduce pain, and improve the quality of life of patients [62-64]. For patients with severe disease, tramadol has been prescribed for a short time. However, there are limitations to its use, such as limited analgesic effect, tolerance, and physical dependence [65]. Acetaminophen is an alternative for patients allergic to NSAIDs, contraindications include liver disease. In studies, acetaminophen is less effective than NSAIDs in the treatment of KOA (SMD 0.05) [66-68].

Surgical treatment. If conservative methods have not shown results for at least three months, surgical intervention should be considered in order to improve the patient's quality of life. Surgical interventions include several methods: arthroscopy, cartilage repair, osteotomy and knee replacement (unicompartmental (UKA) and total knee arthroplasty (TKA)).

Arthroscopy, debridement and osteotomy. Arthroscopic lavage and debridement provide good results in early and moderate KOA, and debridement can relieve symptoms for up to 2 years without increasing the risk of joint arthroplasty [69]. However, the results are notable for their inconsistency [70,71]. The resulting microfractures can positively influence the course of the disease by recruiting bone marrow pluripotent stromal cells to cartilage defects [72]. The traditional Coventry osteotomy transfers weight off the injured area by moderately overcorrecting the varus or valgus axis to relieve pain and slow degenerative process and joint replacement. This intervention is more likely to be performed in younger patients [73,74].

Unicompartmental knee arthroplasty. Single-piece knee replacement is an TKA alternative for patients whose disease is limited to one area of the knee, for example, particularly in the tibiofemoral region (medial or lateral). Being a less invasive procedure, patients can generally be discharged earlier than those undergoing TKA and can return to normal activities earlier, including work and sports [75,76]. The longevity of the medial UKA is ~10 years [77,78].

Total knee arthroplasty. For patients with advanced osteoarthritis of the knee, TKA may be the only option for managing pain and improving function. It is a safe intervention that relieves pain by 90-95% and has an 1-2% complication rate. As surgical techniques have become advanced, more than 90% of TKA patients will continue to report satisfactory outcomes 20 years after surgery [79]. Total knee arthroplasty involves cutting the damaged ends of the tibia and femur and closing them with a prosthesis. Partial recovery takes 6 weeks and full recovery takes up to

Conclusions

Conservative treatment is recommended at the initial stages of KOA, while surgical treatment is indicated for degenerative changes and collapse, which includes arthroscopy, debridement, osteotomy and, ultimately, unicompartmental knee replacement or total knee replacement. In addition, it is necessary to note one of the methods with a potential treatment option – cell therapy, the

1 year [80]. Although the overall life of a TKA prosthesis is affected by a factor associated with orthopedic technologies. In general, the average life is 15–20 years. Survival rates up to 98% have been noted as a long-term outcome of TKA [81]. Postoperative pain appears or persists in one in eight patients without radiological or clinical abnormalities [82]. Infections, femoral-patellar problems, knee stiffness are most common adverse effects. Blacks and Hispanics also have a higher risk of adverse outcomes, including joint infections after TKA [83].

Cell therapy. Current methods of managing articular cartilage defects include multiple microfractures, osteochondral autograft transfer, osteochondral allograft transplantation, autologous chondrocyte implantation, and matrix-assisted autologous chondrocyte implantation [84]. However, these clinical repair methods mainly lead to short-term functional regeneration with the formation of fibrocartilage, and cannot provide sustainable restoration of functional hyaline cartilage [85]. Currently, tissue engineering technology using various hydrogel scaffolds, mesenchymal stem cells (MSCs), and growth factors are considered as the most promising therapeutic strategy for the regeneration of cartilage and osteochondral defects [86]. MSCs are multipotent stem cells that possess self-renewal capacity and can differentiate into various specialized cell types such as adipocytes, chondrocytes, and osteoblasts [87]. MSCs can be obtained from various pathways, such as bone marrow [88], adipose tissue [89], and umbilical cord [90], and have recently been obtained from infrapatellar adipose tissue (IFP) for cartilage regeneration due to their increased chondrogenic capacity [91,92]. One of the advantages of synovium-derived MSCs (SDMSCs) is that these cells are tissue-resident stem cells, which actively participate in maintaining joint homeostasis and cartilage repair [93]. Moreover, it has been demonstrated that compared to MSCs isolated from other tissue sources, SDMSCs have greater proliferation activity and chondrogenic potential in vitro, rendering SDMSCs as an appropriate source for cartilage regeneration [94].

It was reported that the implantation of MSCs alone often leads to the formation of fibrocartilage, indicating that the in vivo environment is not sufficient to induce chondrogenesis in cartilage defect [95]. Recently, a number of studies demonstrated that MSCs encapsulated in hydrogel scaffolds with chondroinductive growth factors significantly repaired cartilage defects in contrast to individual application of MSCs or growth factors [96]. Transplanted MSCs can differentiate directly into chondrocytes and promote cartilage regeneration, secrete a number of growth factors and cytokines, where PDGF is the most effective growth factor and can promote tissue integration [97,98]. There have been many studies on the treatment of KOA [99-101]. MSCs have unique immunomodulatory properties to reduce inflammation, enhancing angiogenesis, cell survival, and differentiation [102,103]. Recent studies have shown that MSC-derived exosomes can inhibit the development of KOA and summarized the results of studies on exosomes derived from various MSCs and their efficacy in KOA therapy [104,105].

purpose of which is to improve the structural integrity of the damaged joint, treat pronounced bone edema and create the potential for remodeling articular cartilage. We hope that such studies would be reasonable, since the most important purpose is to restore the structure of articular cartilage. **Conflict of interest**. The authors declare no conflicts of interest.

Financing. No funding is declared.

Authors' contributions. Conceptualization - J.M.,

B.A.; methodology - D.Sh., B.A.; verification - J.M.; formal analysis - B.A., N.G., D.Sh., J.M.; writing (original draft preparation) - N.G.; writing (review and editing) - B.A., N.G., D.Sh., J.M.

References

1. Zhang Y., Jordan J.M. Epidemiology of Osteoarthritis. Clin Geriatr Med. 2010; 26(3): 355–369. [Crossref]

2. Bortoluzzi A., Furini F., Scirè C.A. Osteoarthritis and its management -Epidemiology, nutritional aspects and environmental factors. Autoimmun Rev. 2018; 17(11): 1097–1104. [Crossref]

3. Mabey T., Honsawek S. Cytokines as biochemical markers for knee osteoarthritis. World J Orthop. 2015; 6(1): 95–105. [Crossref]. https://doi.org/10.5312/wjo.v6.i1.95

4. Nelson A.E. Osteoarthritis year in review 2017: Clinical. Osteoarthr Cartil. 2018; 26(3): 319–325. [Crossref]

5. Martel-Pelletier J., Barr A.J., Cicuttini F.M., Conaghan P.G. et al. Osteoarthritis. Nat Rev Dis Prim. 2016; 2: 1–18. [Crossref] 6. Mobasheri A., Batt M. An update on the pathophysiology of osteoarthritis. Ann Phys Rehabil Med. 2016; 59(5-6): 333-9. [Crossref]

7. Carlson A.K., Rawle R.A., Wallace C.W., Brooks E.G. et al. Characterization of synovial fluid metabolomic phenotypes of cartilage morphological changes associated with osteoarthritis. Osteoarthr Cartil. 2019; 27(8): 1174–1184. [Crossref]

8. GBD 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: A systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016; 388(10053): 1545–1602. [Crossref]

9. de Souza D.V., Lasmar dos Santos M., de Abreu Rodrigues K., Ramires J.B. et al. Exercise and osteoarthrosis: a systematic review. Fisioter Mov. 2013; 26(1): 193–202. [Crossref]

10. Lim J.Y., Tchai E., Jang S.N. Effectiveness of aquatic exercise for obese patients with knee osteoarthritis: a randomized controlled trial. Am J Phys Med Rehabil. 2010; 2(8): 723–31. [Crossref]

11. Emery C.A., Whittaker J.L., Mahmoudian A., Lohmander L.S. et al. Establishing outcome measures in early knee osteoarthritis. Nat Rev Rheumatol. 2019; 15(7): 438–448. [Crossref]

12. Malfait A.M. Osteoarthritis year in review 2015: Biology. Osteoarthritis Cartilage. 2016; 24(1): 21–6. [Crossref]

13. Orlowsky E.W., Kraus V.B. The role of innate immunity in osteoarthritis: When our first line of defense goes on the offensive. J Rheumatol. 2015; 42(3): 363–71. [Crossref]

14. Varady N.H., Grodzinsky A.J. Osteoarthritis year in review 2015: Mechanics. Osteoarthritis Cartilage. 2016; 24(1): 27–35. [Crossref]

15. Arden N., Nevitt M.C. Osteoarthritis: Epidemiology. Best Pract Res Clin Rheumatol. 2006; 20(1): 3–25. [Crossref]

16. Wluka A.E., Lombard C.B., Cicuttini F.M. Tackling obesity in knee osteoarthritis. Nat Rev Rheumatol. 2013; 9(4): 225–35. [Crossref]

17. Mahir L., Belhaj K., Zahi S., Azanmasso H. et al. Impact of knee osteoarthritis on the quality of life. Ann Phys Rehabil Med. 2016; 59(Suppl): e159. [Crossref].

18. Heidari B. Knee osteoarthritis prevalence, risk factors, pathogenesis and features: Part I. Casp J Intern Med. 2011; 2(2): 205–212. [Google Scholar]

19. Hunter D.J., Bierma-Zeinstra S. Osteoarthritis. Lancet. 2019; 393(10182): 1745–1759. [Crossref]

20. Berenbaum F, Wallace I.J., Lieberman D.E., Felson D.T. Modern-day environmental factors in the pathogenesis of osteoarthritis. Nat Rev Rheumatol. 2018; 14: 674–681. [Crossref]

21. Reynard L.N., Barter M.J. Osteoarthritis year in review 2019: Genetics, genomics and epigenetics. Osteoarthr Cartil. 2020; 28(3): 275–284. [Crossref]

22. Rice S.J., Beier F., Young D.A., Loughlin J. Interplay between genetics and epigenetics in osteoarthritis. Nat Rev Rheumatol. 2020; 16(5): 268–281. [Crossref]

23. Wang Y., Meng F., Wu J., Long H. et al. Associations between adipokines gene polymorphisms and knee osteoarthritis: a meta-analysis. BMC musculoskeletal disorders. 2022; 23(1): 166. [Crossref]

24. Workgroup of the American Association of Hip and Knee Surgeons Evidence Based Committee. Obesity and total joint arthroplasty: A literature based review. J Arthroplasty. 2013; 28(5): 714–21. [Crossref]

25. Collins J.E., Katz J.N., Dervan E.E., Losina E. Trajectories and risk profiles of pain in persons with radiographic, symptomatic knee osteoarthritis: data from the osteoarthritis initiative. Osteoarthritis Cartilage. 2014; 22(5): 622–630. [Crossref]

26. Duncan R., Peat G., Thomas E., Wood L. et al. Does isolated patellofemoral osteoarthritis matter? Osteoarthritis Cartilage. 2009; 17(9): 1151–1155. [Crossref]

27. Kellgren J.H., Lawrence J.S. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957; 16(4): 494–502. [Crossref]

28. Bellamy N., Buchanan W.W., Goldsmith C.H., Campbell J. et al. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. J Rheumatol. 1988; 15(12): 1833-1840. [Google Scholar]

29. Hayes M.S., Patterson D.G. Experimental development of the graphic rating method. Psychol Bull. 1921; 18: 98–99. [Crossref]

30. Ayhan E., Kesmezacar H., Akgun I. Intraarticular injections (corticosteroid, hyaluronic acid, platelet rich plasma) for the knee osteoarthritis. World J Orthop. 2014; 5(3): 351–361. [Crossref]

31. Kolasinski S.L., Neogi T., Hochberg M.C., Oatis C. et al. 2019 American College of Rheumatology. Arthritis Foundation Guideline for the Management of Osteoarthritis of the Hand, Hip, and Knee. Arthritis Rheumatol. 2020; 72(2): 220–233. [Crossref]

32. Mora J.C., Przkora R., Cruz-Almeida Y. Knee osteoarthritis: Pathophysiology and current treatment modalities. J Pain Res. 2018; 11: 2189–2196. [Crossref]

33. Rozental T.D., Sculco T.P. Intra-articular corticosteroids: An updated overview. Am J Orthop. 2000; 29(1): 18–23. [Google Scholar]

34. Deyle G.D., Allen C.S., Allison S.C., Gill N.W. et al. Physical Therapy versus Glucocorticoid Injection for Osteoarthritis of the Knee. The New England journal of medicine. 2020; 382(15): 1420–1429. [Crossref]

35. Conaghan P.G., Hunter D.J., Cohen S.B., Kraus V.B. et al. Effects of a Single Intra-Articular Injection of a Microsphere Formulation of Triamcinolone Acetonide on Knee Osteoarthritis Pain: A Double-Blinded, Randomized, Placebo-Controlled, Multinational Study. J Bone Joint Surg Am. 2018; 100(8): 666–677. [Crossref]

36. Saltychev M., Mattie R., McCormick Z., Laimi K. The magnitude and duration of the effect of intra-articular corticosteroid injections on pain severity in knee osteoarthritis. American Journal of Physical Medicine & Rehabilitation. 2020; 99(7): 617–625. [Crossref]

37. Conaghan P.G., Cohen S.B., Berenbaum F., Lufkin J. et al. Brief report: a phase II b trial of a novel extended-release microsphere formulation of triamcinolone acetonide for intraarticular injection in knee osteoarthritis. Arthritis & Rheumatology. 2018; 70(2): 204–211. [Crossref]

38. Klocke R., Levasseur K., Kitas G.D., Smith J.P. et al. Cartilage turnover and intra-articular corticosteroid injections in knee osteoarthritis. Rheumatology International. 2018; 38(8): 455–459. [Crossref]

39. Kapugi M., Cunningham K. Corticosteroids. Orthop Nurs. 2019; 38(5): 336-339. [Crossref]

40. Raeissadat S.A., Gharooee Ahangar A., Rayegani S.M., Minator Sajjadi M. et al. Platelet-Rich Plasma-Derived Growth Factor vs Hyaluronic Acid Injection in the Individuals with Knee Osteoarthritis: A One Year Randomized Clinical Trial. J Pain Res. 2020; 13: 1699-1711. [Crossref]

41. Hacken B., Rogers A., Chinchilli V., Silvis M. et al. Improvement in knee osteoarthritis pain and function following bariatric surgery: 5 year follow-up. Surgery for Obesity and Related Diseases. 2019; 15(6): 979–984. [Crossref]

42. Messier S.P., Gutekunst D.J., Davis C., DeVita P. Weight loss reduces knee-joint loads in overweight and obese older adults with knee osteoarthritis. Arthritis Rheum. 2005; 52(7): 2026–2032. [Crossref]

43. Basu A., Schell J., Scofield R.H. Dietary fruits and arthritis. Food & Function. 2018; 9(1): 70–77. [Crossref]

44. Kovar P.A., Allegrante J.P., MacKenzie C.R., Peterson M.G.E. et al. Supervised Fitness Walking in Patients with Osteoarthritis of the Knee. Annals of internal medicine. 1992; 116(7): 529–534. [Crossref]

45. Segal N.A., Glass N.A., Felson D.T., Hurley M. et al. Effect of quadriceps strength and proprioception on risk for knee osteoarthritis. Med Sci Sports Exerc. 2010; 42(11): 2081–2088. [Crossref]

46. Regnaux J.P., Lefevre-Colau M.M., Trinquart L., Nguyen C. et al. High-intensity versus low-intensity physical activity or exercise in people with hip or knee osteoarthritis. Cochrane Database Syst Rev. 2015; 2015(10): CD010203. [Crossref]

47. Bennell K.L., Lawford B.J., Keating C., Brown C. et al. Comparing Video-Based, Telehealth-Delivered Exercise and Weight Loss Programs With Online Education on Outcomes of Knee Osteoarthritis: A Randomized Trial. Ann Intern Med. 2022; 175(2): 198-209. [Crossref]

48. Phillips S., Li C.S., Phillips M., Bischoff M. et al. Treatment of osteoarthritis of the knee with bracing: A scoping review. Orthop Rev (Pavia). 2016; 8(2): 6256. [Crossref]

49. Fransen M., McConnell S., Harmer A.R., Van der Esch M. et al. Exercise for osteoarthritis of the knee. Cochrane Database Syst Rev. 2015; 2015(1): CD004376. [Crossref]

50. Duivenvoorden T., Brouwer R.W., van Raaij T.M., Verhagen A.P. et al. Braces and orthoses for treating osteoarthritis of the knee. Cochrane Database Syst Rev. 2015; 16(3): CD004020. [Crossref]

51. Mont M.A., Cherian J.J., Bhave A., Starr R. et al. Unloader bracing for knee osteoarthritis: A pilot study of gait and function. Surg Technol Int. 2015; 27: 287–93. [Google Scholar]

52. Reichenbach S., Felson D.T., Hincapié C.A., Heldner S. et al. Effect of Biomechanical Footwear on Knee Pain in People With Knee Osteoarthritis: The BIOTOK Randomized Clinical Trial. JAMA. 2020; 323(18): 1802–1812. [Crossref]

53. Perlman A., Fogerite S.G., Glass O., Bechard E. et al. Efficacy and Safety of Massage for Osteoarthritis of the Knee: a Randomized Clinical Trial. Journal of general internal medicine. 2019; 34(3): 379–386. [Crossref]

54. Tsokanos A., Livieratou E., Billis E., Tsekoura M. et al. The Efficacy of Manual Therapy in Patients with Knee Osteoarthritis: A Systematic Review. Medicina. 2021: 57(7): 696. [Crossref]

55. Bruyère O., Altman R.D., Reginster J.Y. Efficacy and safety of glucosamine sulfate in the management of osteoarthritis: evidence from real-life setting trials and surveys. Seminars in Arthritis and Rheumatism. 2016; 45(4 Suppl): S12–S17. [Crossref]

56. Garcia Rodríguez L.A., Hernández-Díaz S. The risk of upper gastrointestinal complications associated with nonsteroidal anti-inflammatory drugs, glucocorticoids, acetaminophen, and combinations of these agents. Arthritis Res. 2001; 3(2): 98–101. [Crossref]

57. Bhala N., Emberson J., Merhi A., Abramson S. et al. Vascular and upper gastrointestinal effects of non-steroidal antiinflammatory drugs: Meta-analyses of individual participant data from randomised trials. Lancet. 2013; 382(9894): 769–779. [Crossref]

58. Coskun Benlidayi I., Gokcen N., Basaran S. Comparative short-term effectiveness of ibuprofen gel and cream phonophoresis in patients with knee osteoarthritis. Rheumatology International. 2018; 38(10): 1927–1932. [Crossref]

59. Derry S., Conaghan P., Da Silva J.A., Wiffen P.J. et al. Topical NSAIDs for chronic musculoskeletal pain in adults. Cochrane Database Syst Rev. 2016; 4(4): Cd007400. [Crossref]

60. Wadsworth L.T., Kent J.D., Holt R.J. Efficacy and safety of diclofenac sodium 2% topical solution for osteoarthritis of the knee: a randomized, double-blind, vehicle-controlled, 4 week study. Current Medical Research and Opinion. 2016; 32(2): 241–250. [Crossref]

61. da Costa B.R., Reichenbach S., Keller N., Nartey L. et al. Effectiveness of non-steroidal anti-inflammatory drugs for the treatment of pain in knee and hip osteoarthritis: A network meta-analysis. Lancet. 2016; 387(10033): 2093–105. [Crossref] 62. Pavelka K., Bruyère O., Cooper C., Kanis J.A. et al. Diacerein: benefits, risks and place in the management of

osteoarthritis. An opinion-based report from the ESCEO. Drugs & Aging. 2017; 33(2): 75–85. [Crossref] 63. Hochberg M.C. Wohlreich M. Gaynor P. Hanna S. et al. Chinically Polovant Outcomes Based on Analysis of Pooled

63. Hochberg M.C., Wohlreich M., Gaynor P., Hanna S. et al. Clinically Relevant Outcomes Based on Analysis of Pooled Data from 2 Trials of Duloxetine in Patients with Knee Osteoarthritis. The Journal of Rheumatology. 2012; 39(2): 352. [Crossref]

64. Osani M.C., Bannuru R.R. Efficacy and safety of duloxetine in osteoarthritis: a systematic review and meta-analysis. Korean J Intern Med. 2019; 34(5): 966–973. [Crossref]

65. Davies J.E. The pharmacological basis of therapeutics. Occup Environ Med. 2007; 64(8): e2. [Crossref]

66. Bannuru R.R., Schmid C.H., Kent D.M., Vaysbrot E.E. et al. Comparative effectiveness of pharmacologic interventions for knee osteoarthritis: a systematic review and network meta-analysis. Annals of internal medicine. 2015; 162(1): 46–54. [Crossref]

67. Leopoldino A.O., Machado G.C., Ferreira P.H., Pinheiro M.B. et al. Paracetamol versus placebo for knee and hip osteoarthritis. The Cochrane database of systematic reviews. 2019; 2(2): Cd013273. [Crossref]

68. Zhu X., Wu D., Sang L., Wang Y. et al. Comparative effectiveness of glucosamine, chondroitin, acetaminophen or celecoxib for the treatment of knee and/or hip osteoarthritis: a network meta-analysis. Clinical and experimental rheumatology. 2018; 36(4): 595–602. [Google Scholar]

69. Su X., Li C., Liao W., Liu J. et al. Comparison of arthroscopic and conservative treatments for knee osteoarthritis: a 5 year retrospective comparative study. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2018; 34(3): 652–659. [Crossref]

70. Zuiderbaan H.A., van der List J.P., Kleeblad L.J., Appelboom P. et al. Modern indications, results, and global trends in the use of unicompartmental knee arthroplasty and high tibial osteotomy in the treatment of isolated medial compartment osteoarthritis. Am J Orthop (Belle Mead NJ). 2016; 45(6): E355–E361. [Google Scholar]

71. Thorlund J.B., Juhl C.B., Roos E.M., Lohmander L.S. Arthroscopic surgery for degenerative knee: Systematic review and meta-analysis of benefits and harms. BMJ. 2015; 350: h2747. [Crossref]

72. Gobbi A., Karnatzikos G., Kumar A. Long-term results after microfracture treatment for full-thickness knee chondral lesions in athletes. Knee Surgery, Sports Traumatology, Arthroscopy. 2014; 22(9): 1986–1996. [Crossref]

73. Chahla J., Dean C.S., Mitchell J.J., Moatshe G. et al. Medial opening wedge proximal tibial osteotomy. Arthrosc Tech. 2016; 5(4): e919-e928. [Crossref]

74. Fujisawa Y., Masuhara K., Shiomi S. The effect of high tibial osteotomy on osteoarthritis of the knee. An arthroscopic study of 54 knee joints. Orthop Clin North Am. 1979; 10(3): 585–608. [Google Scholar]

75. Rodriguez-Merchan E.C., Gomez-Cardero P. Unicompartmental knee arthroplasty: Current indications, technical issues and results. Efort Open Rev. 2018; 3(6): 363–373. [Crossref]

76. Borus T., Thornhill T. Unicompartmental knee arthroplasty. J Am Acad Orthop Surg. 2008; 16(1): 9-18. [Crossref]

77. Koskinen E., Paavolainen P., Eskelinen A., Pulkkinen P. et al. Unicondylar knee replacement for primary osteoarthritis: A prospective follow-up study of 1,819 patients from the Finnish Arthroplasty Register. Acta Orthop. 2007; 78(1): 128–135. [Crossref]

78. Svärd U.C., Price A.J. Oxford medial unicompartmental knee arthroplasty. A survival analysis of an independent series. J Bone Joint Surg Br. 2001; 83(2): 191–194. [Crossref]

79. Carr A.J., Robertsson O., Graves S., Price A.J. et al. Knee replacement. Lancet. 2012; 379(9823): 1331-40. [Crossref]

80. Leopold S.S. Minimally invasive total knee arthroplasty for osteoarthritis. N Engl J Med. 2009; 360(17): 1749–1758. [Crossref]

81. Keating E.M., Meding J.B., Faris P.M., Ritter M.A. Long-term followup of nonmodular total knee replacements. Clin Orthop Relat Res. 2002; 404: 34–39. [Crossref]

82. Lundblad H., Kreicbergs A., Jansson K.A. Prediction of persistent pain after total knee replacement for osteoarthritis. J Bone Joint Surg Br. 2008; 90(2): 166–171. [Crossref]

83. Nwachukwu B.U., Kenny A.D., Losina E., Chibnik L.B. et al. Complications for Racial and Ethnic Minority Groups After Total Hip and Knee Replacement: A Review of the Literature. JBJS. 2010; 92(2): 338-45. [Crossref]

84. Evenbratt H., Andreasson L., Bicknell V., Brittberg M. et al. Insights into the present and future of cartilage regeneration and joint repair. Cell Regen. 2022; 11(1): 3. [Crossref]

85. Steinert A.F., Ghivizzani S.C., Rethwilm A., Tuan R.S. et al. Major biological obstacles for persistent cell-based regeneration of articular cartilage. Arthritis Res Ther. 2007; 9(3): 213. [Crossref]

86. Huselstein C., Li Y., He X. Mesenchymal stem cells for cartilage engineering. Biomed Mater Eng. 2012; 22(1-3): 69-80. [Crossref]

87. Ding D.C., Shyu W.C., Lin S.Z. Mesenchymal stem cells. Cell Transplant. 2011; 20(1): 5-14. [Crossref]

88. Gnecchi M., Melo L.G. Bone marrow-derived mesenchymal stem cells: Isolation, expansion, characterization, viral transduction, and production of conditioned medium. Methods Mo Biol. 2009; 482: 281–294. [Crossref]

89. Gruber H.E., Deepe R., Hoelscher G.L., Ingram J.A. et al. Human adipose-derived mesenchymal stem cells: Direction to a phenotype sharing similarities with the disc, gene expression profiling, and coculture with human annulus cells. Tissue Eng Part A. 2010; 16(9): 2843-60. [Crossref]

90. Ishige I., Nagamura-Inoue T., Honda M., Harnprasopwat R. et al. Comparison of mesenchymal stem cells derived from arterial, venous, and Wharton's jelly explants of human umbilical cord. Int J Hematol. 2009; 90(2): 261–269. [Crossref]

91. do Amaral R., Almeida H.V., Kelly D.J., O'Brien F.J. et al. Infrapatellar Fat Pad Stem Cells: From Developmental Biology to Cell Therapy. Stem Cells Int. 2017; 2017: 6843727. [Crossref]

92. Stocco E., Barbon S., Piccione M., Belluzzi E. et al. Infrapatellar Fat Pad Stem Cells Responsiveness to Microenvironment in Osteoarthritis: From Morphology to Function. Front Cell Dev Biol. 2019; 7: 323. [Crossref]

93. Koga H., Muneta T., Ju Y.J., Nagase T. et al. Synovial stem cells are regionally specified according to local microenvironments after implantation for cartilage regeneration. Stem Cells. 2007; 25(3): 689-696. [Crossref]

94. Koga H., Muneta T., Nagase T., Nagase T. et al. Comparison of mesenchymal tissues-derived stem cells for in vivo chondrogenesis: suitable conditions for cell therapy of cartilage defects in rabbit. Cell Tissue Res. 2008; 333(2): 207-215. [Crossref]

95. Magne D., Vinatier C., Julien M., Weiss P. et al. Mesenchymal stem cell therapy to rebuild cartilage. Trends Mol Med. 2005; 11(11): 519-526. [Crossref]

96. Wagenbrenner M., Mayer-Wagner S., Rudert M., Holzapfel B.M. et al. Combinations of Hydrogels and Mesenchymal

Stromal Cells (MSCs) for Cartilage Tissue Engineering-A Review of the Literature. Gels. 2021; 7(4): 217. [Crossref]

97. Emadedin M., Ghorbani Liastani M., Fazeli R., Mohseni F. et al. Long-term follow-up of intra-articular injection of autologous mesenchymal stem cells in patients with knee, ankle, or hip osteoarthritis. Archives of Iranian Medicine. 2015; 18(6): 336–344. [Google Scholar]

98. Horie M., Sekiya I., Muneta T., Ichinose S. et al. Intra-articular injected synovial stem cells differentiate into meniscal cells directly and promote meniscal regeneration without mobilization to distant organs in rat massive meniscal defect. Stem Cells. 2009; 27(4): 878–887. [Crossref]

99. Shariatzadeh M., Song J., Wilson S.L. The efficacy of different sources of mesenchymal stem cells for the treatment of knee osteoarthritis. Cell Tissue Res. 2019; 378(3): 399–410. [Crossref]

100. Iijima H., Isho T., Kuroki H., Takahashi M. et al. Effectiveness of mesenchymal stem cells for treating patients with knee osteoarthritis: A meta-analysis toward the establishment of effective regenerative rehabilitation. NPJ Regen Med. 2018; 3: 15. [Crossref]

101. Wang A.T., Feng Y., Jia H.H., Zhao M. et al. Application of mesenchymal stem cell therapy for the treatment of osteoarthritis of the knee: A concise review. World J Stem Cells. 2019; 11(4): 222–235. [Crossref]

102. Pittenger M.F., Discher D.E., Peault B.M., Phinney D.G. et al. Mesenchymal stem cell perspective: Cell biology to clinical progress. NPJ Regen Med. 2019; 4(4): 22. [Crossref]

103. Wang M., Yuan Q., Xie L. Mesenchymal Stem Cell-Based Immunomodulation: Properties and Clinical Application. Stem Cells Int. 2018; 2018: 3057624. [Crossref]

104. Cosenza S., Ruiz M., Toupet K., Jorgensen C. et al. Mesenchymal stem cells derived exosomes and microparticles protect cartilage and bone from degradation in osteoarthritis. Sci Rep. 2017; 7(1): 16214. [Crossref]

105. Ha D.H., Kim H.K., Lee J., Kwon H.H. et al. Mesenchymal Stem/Stromal Cell-Derived Exosomes for Immunomodulatory Therapeutics and Skin Regeneration. Cells. 2020; 9(5): 1157. [Crossref]

Тізе буыны остеоартрозының диагностикасы және емі: Заманауи аспектілері

Байбусунова А.Ж.¹, Нысанбай Г. Н.², Шварц Д.В.³, Жакеева М.Х.⁴

¹ Медицина мектебінің студенті, Астана медицина университеті, Астана, Қазақстан. Е-таіl: baibusunova@icloud.com

² Медицина мектебінің студенті, Астана медицина университеті, Астана, Қазақстан. E-mail: n.n.gauhar@gmail.com ³ Травматология және ортопедия кафедрасының ассистенті, Астана медицина университеті, Астана, Қазақстан. E-mail: shwartz_dima81@mail.ru

⁴ Нефрология, гематология, аллергология және иммунология курстары бар ішкі аурулар кафедрасының ассистенті, Астана медицина университеті, Астана, Қазақстан. Е-mail: mikena90@mail.ru

Түйіндіме

Тізе буыны остеоартрозы (ТБОА) - жиі жағдайда 60 жастан асқан ересектерге әсер ететін созылмалы буын ауруы. Соңғы зерттеулер оның прогрессивті сипатын көрсетеді, буынның әртүрлі компоненттеріндегі құрылымдық өзгерістерді, соның ішінде буын шеміршегін, синовиальды қабықты және байламдарды қамтиды.

Жаһандық деңгейде 250 миллионға жуық адам ТБОА-мен ауырады, симптомсыз жағдайларға, семіздікке және басқа факторларға байланысты ТБОА жиілігі арта түскен. Еркектерге қарағанда әйелдер бұл ауруға жиі ұшырайды. ТБОА әлемдегі мүгедектіктің жиі кездесетін себептерінің біріне айналып, аурудың ауыртпалығының 85%-ын құрайды. 60 жастан асқан адамдардың шамамен 10%-ы ТБОА-мен ауырады, бұл қозғалыс шектеулеріне әкеледі.

Шолудың мақсаты ТБОА-ны бейнелеу мен заманауи емдеуге қатысты соңғы зерттеулерді, сондай-ақ осы аурудың таралуын талдауды қамту болып табылды.

Процестің ауырлығына және ауырсыну синдромына байланысты хирургиялық емес әдістер жиі жағдайда жеткіліксіз болып жатады. Буындарды эндопротездеу соңғы сатыда емдеудің маңызды әдісін ұсынады. Алайда, кортикостероидты инъекциялар, гиалурон қышқылы және жасушаларды қолдану сияқты басқа әдістер аурудың дамуын бәсеңдетуі мүмкін. Ерте кезеңдерде ТБОА асимптоматикалық болуы мүмкін екенін ескере отырып, уақтылы диагноз қою маңызды рөл атқарады. Ауруды тиімді басқару үшін симптомдарды, клиникалық деректерді және әртүрлі диагностикалық бейнелеу әдістерінің нәтижелерін ескеру маңызды.

Түйін сөздер: тізе буыны остеоартрозы, остеоартроздың диагностикасы, остеоартроздың емі, толық эндопротездеу, мезенхималық бағаналы жасушалар.

Диагностика и лечение остеоартроза коленного сустава: Современные аспекты

Байбусунова А.Ж.¹, Нысанбай Г. Н.², Шварц Д.В.³, Жакеева М.Х.⁴

¹ Студент Школы медицины, Медицинский университет Астана, Астана, Казахстан. E-mail: baibusunova@icloud.com

² Студент Школы медицины, Медицинский университет Астана, Астана, Казахстан. E-mail: n.n.gauhar@gmail.com ³ Ассистент кафедры травматологии и ортопедии, Медицинский университет Астана, Астана, Казахстан.

E-mail: shwartz_dima81@mail.ru

⁴ Ассистент кафедры внутренних болезней с курсами нефрологии, гематологии, аллергологии и иммунологии, Медицинский университет Астана, Астана, Казахстан, mikena90@mail.ru

Резюме

Остеоартроз коленного сустава (ОАКС) представляет собой распространенное хроническое заболевание суставов, чаще всего поражающее взрослых старше 60 лет. Последние исследования указывают на его прогрессивный характер, вовлекая структурные изменения в различных компонентах сустава, включая суставной хрящ, синовиальную оболочку и связки.

Глобально около 250 миллионов человек страдают от ОАКС, с ростом заболеваемости ОАКС из-за бессимптомных случаев, ожирения и других факторов. Женщины чаще подвержены этому заболеванию, чем мужчины. ОАКС становится одной из главных причин инвалидности в мире, обуславливая 85% бремени ОАКС. Приблизительно 10% людей старше 60 лет страдают от ОАКС, что приводит к ограничениям в передвижении. В связи с выраженностью процесса и болевого синдрома, нехирургические методы часто оказываются недостаточными.

Цель обзора заключается в освещении недавних исследований, касающихся визуализации и современных методов лечения ОАКС, а также анализа распространенности этого заболевания.

Эндопротезирование суставов представляет собой важный метод лечения на терминальной стадии. Однако другие методы, такие как инъекции кортикостероидов, гиалуроновая кислота и использование клеток, могут замедлить прогрессирование заболевания. Своевременная диагностика играет ключевую роль, учитывая, что на ранних стадиях ОАКС может протекать бессимптомно. Важно учитывать симптомы, клинические данные и результаты различных методов визуализации для эффективного управления заболеванием.

Ключевые слова: остеоартроз коленного сустава, диагностика остеоартроза, лечение остеоартроза, тотальное эндопротезирование, мезенхимальные стволовые клетки.