JASIŃSKI, Karol, DUBNIEWICZ, Ewa, MAJEWSKA, Paula, STASZEK, Alicja, ŁOSKOT, Wiktoria, SZWECH, Jan, MATCZAK, Mateusz, BRODA, Aleksandra, HOKSA, Kacper and JODŁOWSKI, Krzysztof. Osteoarthritis in obesity - Summary. Journal of Education, Health and Sport. 2025;79:57898. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.79.57898
https://apcz.umk.pl/JEHS/article/view/57898

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 17.01.2025. Revised: 02.03.2025. Accepted: 02.03.2025. Published: 05.03.2025.

Osteoarthritis in obesity - Summary

AUTHORS

Karol Jasiński

Provincial Hospital of St. Luke in Tarnow, ul. Lwowska 178a, 33-100 Tarnow

ORCID: https://orcid.org/0009-0004-6845-5199

E-mail: karol.jasinski99@gmail.com

Ewa Dubniewicz

Central Clinical Hospital of Medical University of Lodz, ul. Pomorska 251, 92-213 Lodz

ORCID: https://orcid.org/0009-0007-4191-6794

E-mail: edubniewicz@gmail.com

Paula Majewska

Central Clinical Hospital of Medical University of Lodz, ul. Pomorska 251, 92-213 Lodz

ORCID: https://orcid.org/0009-0003-7934-397X

E-mail: majewskapaula14@gmail.com

Alicja Staszek

Central Clinical Hospital of Medical University of Lodz, ul. Pomorska 251, 92-213 Lodz

ORCID: https://orcid.org/0009-0007-0323-8697

E-mail: staszekalicja0@gmail.com

Wiktoria Łoskot
University Clinical Hospital No. 2 of the Medical University of Lodz: Łódź, ul. Stefana Żeromskiego 113, 90-549 Łódź, PL
ORCID: https://orcid.org/0009-0001-5392-4835
E-mail: wiktoria.loskot98@gmail.com

Jan Szwech
Masovian Specialist Hospital, ul. Jana Aleksandrowicza 5, 26-617 Radom
ORCID: https://orcid.org/0009-0002-9312-8152
E-mail: szwechjan@gmail.com

Mateusz Matczak
Hospital of the Ministry of Interior and Administration in Lodz, ul. Północna 42, 91–425 Łódź
ORCID: https://orcid.org/0009-0000-9701-406X
E-mail: matczak.mateusz@icloud.com

Aleksandra Broda
Hospital of the Ministry of Interior and Administration in Lodz, ul. Północna 42, 91–425 Łódź
ORCID: https://orcid.org/0009-0004-5179-9411
E-mail: aleksandra.d.broda@gmail.com

Kacper Hoksa
Hospital of the Ministry of Interior and Administration in Lodz, ul. Północna 42, 91–425 Łódź
ORCID: https://orcid.org/0009-0007-9832-7093
E-mail: kacper.hoksa98@gmail.com

Krzysztof Jodłowski
Hospital of the Ministry of Interior and Administration in Lodz, ul. Północna 42, 91–425 Łódź
ORCID: https://orcid.org/0009-0003-9041-2091
E-mail: lek.krzysztofjodlowski@gmail.com

Abstract

Osteoarthritis (OA) is a slowly progressive joint disease that primarily affects articular cartilage and gradually causes pain, stiffness, and immobility. This problem negatively affects the quality of life of a large part of the population and consumes a significant part of the state budget. It ranks 11th in the list

of global health issues in relation to disability. Primary OA occurs due to factors such as age progression, obesity, joint injuries, and anatomical features. Secondary OA frequently associates with other pathologies such as Paget's disease and osteoporosis. Of all the risk factors for KOA (knee osteoarthritis), obesity is the most critical since increasing body weight increases joint stress and hastens cartilage degradation. Weight loss, physical activity, management of symptoms forms in prevention and the management of KOA. Interventions of NSAIDs, glucocorticoids injections, and physiotherapy may help. Total knee arthroplasty remains the most effective treatment for patients with end-stage OA.

Keywords: osteoarthritis, knee osteoarthritis, obesity and osteoarthritis, risk factors of osteoarthritis, knee joint degeneration

1. Osteoarthritis: Pathophysiology, Risk Factors, and Global Impact

Osteoarthritis (OA) is a continual disease that affects joints and tissues. Articular cartilage is the first to be damaged in this condition. Less often bone tissue under the cartilage is undergoing remodeling, other synovial structures around are ran-down and osteophytes can be formed [2,3].

To the main manifestations of OA belongs pain, stiffness, swelling and limited mobility in joints. Apart from difficulties in motility, chronic pain can impair mental condition [3].

In 2017, OA affected 3754,2 out of 100 000 adult people [4], furthermore OA is in 11th place among contributors to global disability according to years lived with disabilities (YLDs) [2].

Considering to causes, OA is divided into primary and secondary.

Primary OA is the most prevalent subset of the disease and is strongly related to the main causes i.e. age, obesity, joint injures, female gender, muscle weakness, anatomical factors.

Traumatic knee injury and obesity are two main factors of knee OA. Additionally, it can be noticed that the risk increases with age. The median age of onset of knee OA is 55 years. [17].

Secondary OA is associated with Paget disease, osteoporosis, osteochondritis, metabolic disorders, Marfan syndrome, Ehlers-Danlos syndrome congenital joints disorders, avascular necrosis [7] and atopic diseases (atopic dermatitis and asthma) [18].

2. Knee osteoarthritis and obesity relationship

Obesity is a problem regarding above 2 billion people all around the world. This group includes persons with BMI above 25. It is worth mentioning that 10% of the 2 billion obese people have a BMI over 30 which particularly increases the risk of obesity-related complications [1].
In the conducted research a correlation between obesity and knee osteoarthritis (KOA) can be seen. In every age group above 25 years old prevalence od symptomatic KOA is higher in obese people than non-obese. In both obese and non-obese people, the greatest increase in symptomatic knee OA occurs between the ages of 55 and 65, but in group of people with increased BMI the growth is greater.
Lifetime risk of symptomatic knee OA in obese female is 28,85% and in obese male is 15,7%.
In non-obese female risk is 12,22% and in non-obese male is 9,6%.
As an example of this study the risk of symptomatic knee OA caused by obesity increases by 16,63% in women and 6,1% in men. Female are more likely to develop symptomatic KOA through obesity than male [17].

3. Impact of obesity on knee-forming components

Knees are one of weight-bearing joints [6]. In obese people with higher body weight, the knees are exposed to much greater loads than in non-obese people. Obesity is considered as a initiating factor of KOA because of excessive mechanical loading of the joint.
Overloading of the joint is underlying cause of the alterations in joint structure that impacts on elements that builds knee joint [8].
Knee is the most often involved in osteoarthritis joint [5]. In this condition all components of knee joint can be alimented i.e. joint cartilage with underlying subchondral bone, synovial stratum, adjoining ligaments and muscles, Hoffa's fat pad. When all mentioned elements are affected, we call it whole joint disease [6].

Articular cartilage destruction is associated with an increased body mass index (BMI). [9] People with increased BMI suffered from more severe cartilage damage [10] and individuals with a higher body mass index had thicker cartilage on the patella and femoral groove, while those with a lower BMI had thinner cartilage on the medial tibia [11].

Bone-cartilage interface on which higher forces act is more susceptible to horizontal fissuring. Horizontal fissures are described as irregular cartilage erosion with fibrogranulation tissue infiltration. There is presence of cartilage/bone debris and the rupture of microcapillaries within the fissures at the osteochondral interface. Formation of this abnormality is crucial in pathogenesis of OA in obese people [12].

Subchondral bone under pressure increased by overweight more often creates osteoid [12]. In the initial stage of knee OA bone remodeling is increased and is the cause of a subchondral bone plate thickness reduction and reduced bone volume in trabecular bone. The number of microcracks can increase, but this process is required for cartilage homeostasis and might regulate subchondral bone remodeling [13].

Synovium in people with increased BMI differs than non-obese people. The difference is that in people without overweight, synovial fluid contains less inflammatory factors such as IL-6, IL-8, TNF-alpha (tumor necrosis factor) [14]. Inflammatory factors leads to synovitis, which is associated with knee joint complainments [15].

Menisci as an important part of the knee joint that bears the weight of the body is exposed to mechanical damage especially in people with higher body weight. Obesity significantly increases risk of meniscal damage [16]. Tears, destruction and extructions are caused by the increased load resulting from increased body weight.

4. Prevention and treatment of knee OA

Increased Body Mass Index (BMI) is a significant factor increasing the risk of knee OA and occurrence of symptoms, therefore reduction of weight should be the first step in both prevention and treatment.

Physical activity on addiction to reducing body weight, also affects on reduction symptoms of knee OA like pain, knee function outcomes, improves quality of life (QoL). The major types of exercises that shows great results are aerobic, isometric and resistance trainings. For best results, exercises should be adapted to the severity of KOA and be performed 3-5 times a week for an hour. High-intensity aquatic resistance training provides significant benefits in managing KOA. It not only enhances fitness but also reduces body mass, a substantial risk factor for KOA development [19]. Strongly recommended for the management of KOA are also Tai chi, cane and tibiofemoral knee braces [21].

Reducing pain is important for continuing exercises for further benefits.

Oral NSAIDs are the primary treatment for osteoarthritis (OA) and are strongly recommended due to their established short-term efficacy. They are the first-line choice for OA management, regardless of the affected joint.

While this guideline does not compare specific NSAIDs, some may have more favorable side effect profiles. Safe use requires careful patient selection, regular monitoring for gastrointestinal, cardiovascular, and renal risks, and awareness of drug interactions. NSAID doses should be kept low and treatment duration as brief as possible. Topical NSAIDs like a diclofenac, ibuprofen, ketoprofen are strongly recommended for patients suffering from KOA. Topical capsaicin can be considered individually [21].

Glucocorticoids in the form of injections into the knee joint are strongly recommended for patients with KOA and demonstrates short-term efficacy [21].

Intra-articular injections of botulinum toxin and hyaluronic acid can be used to relieve acute pain. It is worth considering their use despite side effects such as infection, bleeding, allergic reactions and rare damage of the joint [20].

Physiotherapy shows little effectiveness in the treatment of rapid pain but plays a significant role in long-term functional recovery [20].

Therapy with mesenchymal stromal cells (MSC) for KOA is under research. There are reports of a reduction in pain compared to placebo using MSC, but the effectiveness of this therapy is still unconfirmed [22].

Total knee arthroplasty in patients with end-stage KOA is the most effective procedure and often performed. It helps fight pain effectively with less use of painkillers, which are associated with more side effects [23].

Conclusions

Knee osteoarthritis is an increasingly observed form of degenerative arthritis, and obesity is among its key risk factors that can be modified. Weight management and physical exercises should be promoted in the prevention and management of symptoms in OA. Early incidence, with proper medication and therapy, increases quality of life significantly. Definitive intervention is available in advanced cases, for example, total knee arthroplasty. Addressing the obesity crisis will contribute to a global reduction in the burden of OA and improve patients' health.

Disclosure

Authors do not report any disclosures

Authors 'contribution:

Conceptualization: Ewa Dubniewicz, Mateusz Matczak, Alicja Staszek, Kacper Hoksa

Methodology: Karol Jasiński, Wiktoria Łoskot, Krzysztof Jodłowski

Investigation: Mateusz Matczak, Paula Majewska, Alicja Staszek, Jan Szwech

Software: Kacper Hoksa, Mateusz Matczak, Aleksandra Broda

Formal analysis: Ewa Dubniewicz, Wiktoria Łoskot, Jan Szwech,

Writing: Karol Jasiński

Resources: Paula Majewska, Kacper Hoksa, Karol Jasiński

Supervision: Krzysztof Jodłowski, Aleksandra Broda, Wiktoria Łoskot, Jan Szwech, Alicja Staszek

All authors have read and agreed with the published version of the manuscript.

Funding statement:

The study did not receive special funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Data Availability Statement:

Not applicable.

Conflict of Interest Statement:

The authors declare no conflict of interest.

References

[1] B. Caballero, *Humans against obesity: who will win?* Adv. Nutr., 10 (suppl_1) (2019), pp. S4-S9, doi: 10.1093/advances/nmy055

[2] R. Giorgino, D. Albano, S. Fusco, et al., Knee osteoarthritis: epidemiology, pathogenesis, and mesenchymal stem cells: what else is new? An update, Int. J. Mol. Sci., 24(7) (2023), p. 6405, doi: 10.3390/ijms24076405

[3] K.D. Allen, L.M. Thoma, Y.M. Golightly, *Epidemiology of osteoarthritis*,doi: 10.1016/j.joca.2021.04.020

[4] S. Safiri, A.-A. Kolahi, E. Smith, et al., Global, regional and national burden of osteoarthritis 1990–2017: A systematic analysis of the Global Burden of Disease Study 2017, Ann. Rheum. Dis., 79 (2020), pp. 819–828, doi: 10.1136/annrheumdis-2020-216942

[5] R. Shumnalieva, G. Kotov, P. Ermencheva, S. Monov, Pathogenic Mechanisms and Therapeutic Approaches in Obesity-Related Knee Osteoarthritis, MDPI, doi: https://doi.org/10.3390/biomedicines12010009

[6] D. Primorac, V. Molnar, E. Rod, et al., Knee Osteoarthritis: A Review of Pathogenesis and State-Of-The-Art Non-Operative Therapeutic Considerations, Genes, 11 (2020), p. 854, doi: 10.3390/genes11070854

[7] R. Sen, J.A. Hurley, *Osteoarthritis, doi:* https://www.ncbi.nlm.nih.gov/books/NBK482326/

[8] L. Chen, J.J.Y. Zheng, G. Li, et al., Pathogenesis and clinical management of obesity-related knee osteoarthritis: Impact of mechanical loading, J. Orthop. Transl., doi: https://doi.org/10.1016/j.jot.2020.05.001

[9] A. Anandacoomarasamy, G. Smith, S. Leibman, et al., *Cartilage defects are associated with physical disability in obese adults*, Rheumatology, 48 (10) (2009), pp. 1290–1293, doi: 10.1093/rheumatology/kep246

[10] C. Ding, F. Cicuttini, F. Scott, et al., *Knee structural alteration and BMI: a cross-sectional study*, Obes. Res., 13 (2) (2005), pp. 350–361, doi: 10.1016/j.jot.2020.05.001

[11] M.R. Widmyer, G.M. Utturkar, H.A. Leddy, et al., High body mass index is associated with increased diurnal strains in the articular cartilage of the knee, Arthritis Rheum., 65 (10) (2013), pp. 2615–2622, doi: 10.1002/art.38062

[12] L. Chen, F. Yao, T. Wang, et al., Horizontal fissuring at the osteochondral interface: a novel and unique pathological feature in patients with obesity-related osteoarthritis, Ann. Rheum. Dis., 79 (2020), pp. 811–818, doi: 10.1136/annrheumdis-2020-216942

[13] M. Zarka, E. Hay, A. Ostertag, et al., Microcracks in subchondral bone plate are linked to less cartilage damage, Bone, 123 (2019), pp. 1–7, doi: 10.1016/j.bone.2019.03.011

[14] M.J. Pearson, D. Herndler-Brandstetter, M.A. Tariq, et al., IL-6 secretion in osteoarthritis patients is mediated by chondrocyte-synovial fibroblast cross-talk and is enhanced by obesity, Sci. Rep., 7 (1) (2017), p. 3451, doi: 10.1038/s41598-017-03759-w

[15] B.J. de Lange-Brokaar, A. Ioan-Facsinay, E. Yusuf, et al., Association of pain in knee osteoarthritis with distinct patterns of synovitis, Arthritis Rheumatol., 67 (3) (2015), pp. 733–740, doi: 10.1002/art.38965

[16] F. Zhang, S.M. Bierma-Zeinstra, E.H.G. Oei, et al., Factors associated with meniscal body extrusion on knee MRI in overweight and obese women, Osteoarthritis Cartilage, 25 (5) (2017), pp. 694–699, doi: 10.1016/j.joca.2016.12.001

[17] E. Losina, A.M. Weinstein, W.M. Reichmann, et al., Lifetime risk and age at diagnosis of symptomatic knee osteoarthritis in the US, Arthritis Care Res., doi: https://acrjournals.onlinelibrary.wiley.com/doi/pdf/10.1002/acr.21898

[18] M.C. Baker, K. Sheth, R. Lu, et al., Increased risk of osteoarthritis in patients with atopic disease, Ann. Rheum. Dis., 2022, doi: 10.1136/ard-2022-223640

[19] A. Canzone, F. Roggio, A. Patti, et al., Classification of physical activity programs based on the Kellgren & Lawrence scale for knee osteoarthritis: A systematic review, MSC, doi: https://doi.org/10.1002/msc.70019

[20] D. Donati, F. Giorgi, T. Domiziano, et al., Maximizing knee OA treatment: A comparative look at physiotherapy and injections, J. Pers. Med., 14(11) (2024), p. 1077, doi: 10.3390/jpm14111077

[21] S.L. Kolasinski, T. Neogi, M.C. Hochberg, et al., 2019 American College of Rheumatology/Arthritis Foundation guideline for the management of osteoarthritis of the hand, hip, and knee, Arthritis Rheumatol., doi: https://doi.org/10.1002/art.41142

[22] C.G. Tabet, R.L. Pacheco, A.L.C. Martimbianco, et al., Advanced therapy with mesenchymal stromal cells for knee osteoarthritis: Systematic review and meta-analysis of randomized controlled trials, J. Orthop. Translat., 48 (2024), pp. 176–189, doi: 10.1016/j.jot.2024.07.012

[23] D.N. de Souza, N.A. Lorentz, L. Charalambous, et al., Comprehensive pain management in total joint arthroplasty: A review of contemporary approaches, J. Clin. Med., 13(22) (2024), p. 6819, doi: 10.3390/jcm13226819

[24] F. Dobson, R. Roos, M. Stratford, et al., Reliability and measurement error of the Osteoarthritis Research Society International (OARSI) recommended performance-based tests of physical function in people with hip and knee osteoarthritis, Osteoarthritis Cartilage, 25(11) (2017), pp. 1792–1796, doi: 10.1016/j.joca.2017.06.006.

[25] A.C. Skelly, R. Chou, J.R. Dettori, et al., Noninvasive nonpharmacological treatment for chronic pain: a systematic review, Agency for Healthcare Research and Quality, 2018, available at: https://effectivehealthcare.ahrq.gov/sites/default/files/pdf/nonpharma-chronic-pain-cer-209.pdf.

[26] S.P. Messier, A.E. Resnik, D.P. Beavers, et al., Intentional weight loss in overweight and obese patients with knee osteoarthritis: is more better?, Arthritis Care Res. (Hoboken), 70 (2018), pp. 1569–1575, doi: 10.1002/acr.23608.

[27] F. Rannou, J.P. Pelletier, J. Martel-Pelletier, Efficacy and safety of topical NSAIDs in the management of osteoarthritis: evidence from real-life setting trials and surveys, Semin. Arthritis Rheum., 45 (2016), Suppl. S18–21, doi: 10.1016/j.semarthrit.2015.11.007.

[28] D.H. Solomon, M.E. Husni, P.A. Libby, et al., The risk of major NSAID toxicity with celecoxib, ibuprofen, or naproxen: a secondary analysis of the PRECISION trial, Am. J. Med., 130 (2017), pp. 1415–1422, doi: 10.1016/j.amjmed.2017.06.028.

[29] A. Hajihasani, M. Rouhani, M. Salavati, et al., The influence of cognitive behavioral therapy on pain, quality of life, and depression in patients receiving physical therapy for chronic low back pain: a systematic review, PM R, 11 (2019), pp. 167–176, doi: 10.1016/j.pmrj.2018.09.029.

[30] F.K. Chan, J.Y. Ching, Y.K. Tse, et al., Gastrointestinal safety of celecoxib versus naproxen in patients with cardiothrombotic diseases and arthritis after upper gastrointestinal bleeding (CONCERN): an industry-independent, double-blind, double-dummy, randomised trial, Lancet, 389 (2017), pp. 2375–2382, doi: 10.1016/S0140-6736(17)30981-9.