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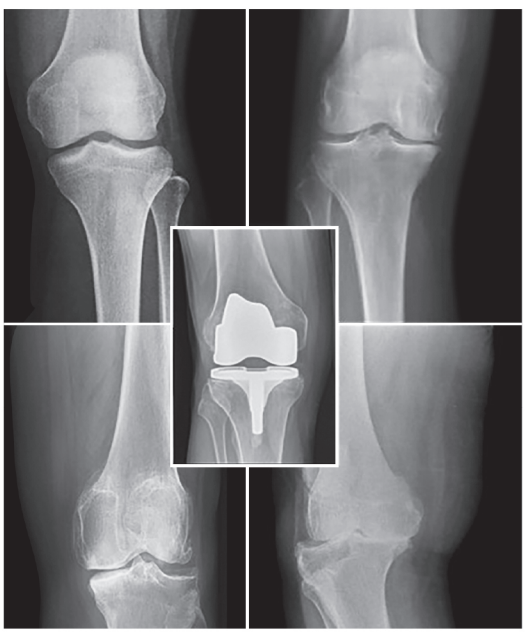
2

Master Series Monographs
Osteoarthritis Knee



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Contents

Note from the Editor-in-Chief xi

Note from the Section Editors xiii

Contributors xv

1. **Applied Anatomy and Biomechanics of the Knee Joint** 1
   Mihir Patel, Vedant Zaveri, Gautam Zaveri

2. **Etiopathogenesis and Natural History of Knee Osteoarthritis** 13
   Abhay Elhence, Vedant Zaveri, Rajesh Rajnish

3. **Clinical Features, Radiology, and Classification of Knee Osteoarthritis** 19
   Vaibhav Bagaria, Vedant Zaveri

4. **Patellofemoral Osteoarthritis** 31
   Arzan Jesia, Clevio Desouza, Rammohan Yedave, Dinshaw Pardiwala

5. **Nonoperative Treatment of Osteoarthritis Knee** 47
   Biswajit Sahu, Binaya Kumar Mishra

6. **Role of Intra-articular Injections and Orthobiologics in Osteoarthritis Knee** 55
   Vijay D Shetty, Clevio Desouza

7. **Knee Preservation Surgery: Role of Arthroscopy in Osteoarthritis and Prevention of Osteoarthritis** 69
   Abhay Narvekar, Gautam Zaveri

8. **Realignment Procedures for Osteoarthritis Knee** 81
   Bhushan M Sabnis

9. **Role of Unicompartmental Knee Replacement in Knee Osteoarthritis** 95
   Samuel King, Hemant Pandit

10. **Role of Total Knee Arthroplasty for Osteoarthritis Knees** 105
    Pranav Keswani, Shubhranshu S Mohanty

11. Osteoarthritis Knee: Case Series **123**
Ameet Pispati, Kedar Aher

Case 1: Stage 0—Prearthritis *125*

Case 2: Stage 1—Minor Osteoarthritis—Early Signs and Gentle Interventions *126*

Case 3: Stage 2—Mild Osteoarthritis—Increasing Discomfort and Functional Impairment *127*

Case 4: Stage 3—Moderate Osteoarthritis—Significant Pain and Functional Impairment *129*

Case 5: Stage 4—Severe Osteoarthritis—Significant Pain and Functional Impairment *130*

Case 6: Medial Compartment Osteoarthritis in Older Patient with Pain and Functional Impairment *131*

Case 7: Medial Compartment Knee Osteoarthritis in Young Active Patient *132*

Index **133**

2 Etiopathogenesis and Natural History of Knee Osteoarthritis

Abhay Elhence, Vedant Zaveri, Rajesh Rajnish

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Introduction

Knee osteoarthritis (OA) is a chronic, slowly progressive, degenerative condition that primarily affects joint cartilage, with secondary involvement of the synovium, menisci, ligaments, muscles, and subchondral bone. The predominant clinical symptoms of knee OA are pain, swelling, and difficulty in walking.

The Global Burden of Disease Study 2015, estimates that upward of 200 million individuals worldwide suffer from knee OA, constituting 85% of all OA cases.[1] The study estimates a rise in the prevalence of knee OA by 32.7% between 2005 and 2015, probably a reflection of the aging global population and increase in the prevalence of risk factors such as obesity.[2] Currently, the prevalence of knee OA in adults over 60 years of age is about 10% in men and 13% in females.[3] The projected lifetime risk of developing symptomatic knee OA is 45%.[4] The management of knee OA places a huge direct and indirect economic burden on the healthcare machinery.[1] Hence, it is vital to understand the natural history and pathogenesis of the disease in order to develop effective nonoperative treatments that slow down the natural progression of OA, and postpone or even replace the need for surgery. This chapter will discuss the etiopathogenesis and natural history of the OA of the knee.

Risk Factors

The development and progression of knee OA is thought to be multifactorial with various modifiable and nonmodifiable risk factors being identified, including genes, age, gender, ethnicity, obesity, metabolic and hormonal factors, joint malalignment, acute trauma, chronic overload, and participation in sports.[5,6]

Genetic predisposition: Genetics is known to have a major influence on the development and progression of OA. More than 90 different genetic risk loci have been identified, although they have small effect sizes.[7] Additionally, mutations in certain genes causing osteoarthritis, could also determine the age of disease onset, joints to be affected, and progression of disease. Studies have demonstrated that 39–78% of OA can be attributed to genetic factors.[8]

Aging: Advancing age is the single biggest risk factor for the development of OA. The Johnson County Osteoarthritis Project in the USA reported that the prevalence of radiographic knee OA rose from 26.2% in the 55–64 years range to nearly half of participants in the ≥ 75 group. The prevalence of symptomatic knee OA likewise increased from 16.3 to 32.8% between these groups.[9] However, Losina et al, pointed out that although overall prevalence rates increased with increasing age, the highest incidence of new symptomatic knee OA was within the 55–64 years age group, with numbers tapering on either side.[10] With aging, chondrocytes show an inability to produce proteoglycans needed to maintain the compressive strength of the cartilage matrix. Hence, there is a decreased threshold for load-induced damage.[11]

Female sex: After the age of 50 years, there is a steep increase in the incidence of knee OA in women compared to men.[12] Women are also more likely to be symptomatic and suffer from more severe arthritis than men.[13] It is postulated that women may be prone to developing OA

due to genetic differences in the meniscus, a higher prevalence of metabolic syndrome, and kinematic differences such as a wider pelvis with a higher Q angle that places greater loads on the lateral compartment of the knee.[14]

Obesity: Obese patients have three times the risk of developing knee OA compared to normal weight controls, making obesity one of the most substantial risk factors for knee OA.[15] Obesity can also cause OA to progress faster. A greater body weight exerts greater loads on the knees, which results in wear and tear of joint cartilage.[16] Obesity and type 2 diabetes can lead to metabolic dysfunction which increases the risk of developing OA.[17] Finally, obesity results in the formation of adipose tissue that secretes adipokines, a biologically active substance that contributes to joint inflammation and alteration in cartilage homeostasis, making the joint more susceptible to OA.[18] The Framingham study suggested that women who decreased their body mass index (BMI) by ≥ 2 units 10 years before the examination had a 50% reduction in the risk of knee OA.[19]

Joint injury: Acute injuries to the knee resulting in intraarticular fractures, ruptured ligaments and meniscal tears can alter joint mechanics, precipitating progressive degeneration of the articular cartilage. Intraarticular fractures resulting in cartilage damage and incongruity of joint surfaces may precipitate early onset OA, even if the joint surfaces are realigned surgically. An isolated rupture of the anterior cruciate ligament (ACL) leads to early onset OA in 13% cases after 10–15 years.[20] This number increases to 21–40% when there is accompanying injury to menisci, collateral ligaments or joint cartilage.[21] Although ACL reconstruction may relieve pain and improve joint function, it may not prevent knee OA in the long-term. Similarly, repair of torn meniscus may relieve pain and improve knee function, but its efficacy in preventing knee OA is unknown. Total meniscectomy has been shown to significantly increase the risk of developing radiographic knee OA.[22]

Chronic mechanical overloading: Occupations that involve repetitive joint use, such as squatting and kneeling may result in mechanically mediated loss of articular cartilage in high-stress areas causing an increase in expression of different cytokines and proteolytic enzymes that may precipitate OA.[11,23] Similarly, high-impact sports like football, hockey, and long-distance running cause increased joint stress and increase the risk of Knee OA.[24]

Malalignment: Although, a dire association between malalignment and the onset of OA has not been firmly established, studies have clearly shown that abnormal alignment of the knee results in accelerated degeneration. Cerejo et al, reported a four-fold higher risk of progressive medial compartment OA in patients with varus alignment, and a five-fold higher risk of lateral compartment OA in those with valgus alignment.[25]

Pathogenesis

Traditionally, OA was thought to be due to wear and tear of the joint with age. However, current research suggests that in addition to mechanical factors, inflammation, and metabolic factors play an important role in the pathogenesis of OA. The development and progression of OA involves a complex interplay between various risk factors in genetically predisposed individuals **(Fig 1)**.[5]

Osteoarthritis begins with failure of cartilage homeostasis which leads to progressive degradation of articular cartilage, synovial inflammation, alteration in the quality, and quantity of synovial fluid and remodeling of subchondral bone. Concomitant structural and functional changes are seen in the meniscus, ligaments, joint capsule, and periarticular muscles **(Flowchart 1)**.[26] Clinically, these lead to pain, stiffness, deformity, functional limitation, and disability.

Aging or oxidative stress can result in changes in the composition and structure of chondrocytes.[5,27] This triggers the secretion of catabolic

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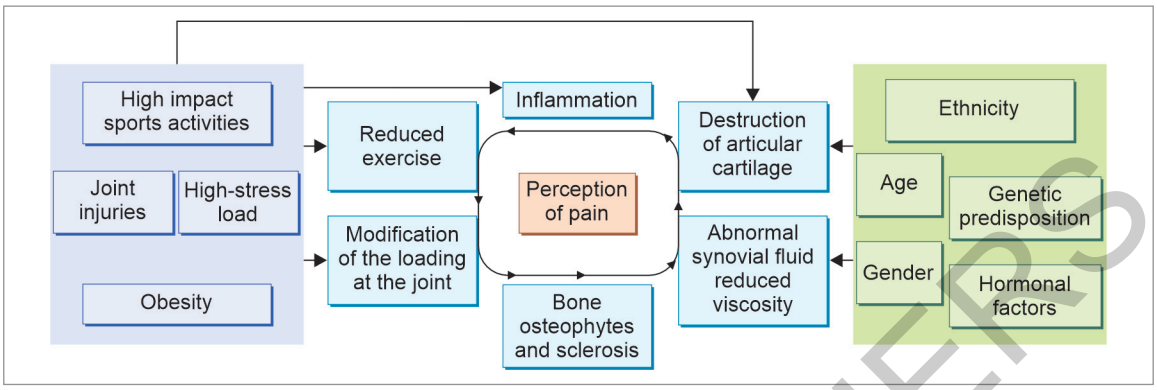
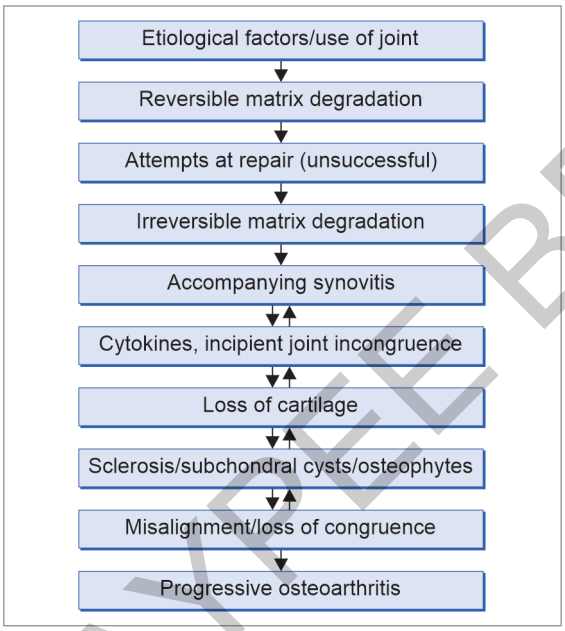


Fig 1: Diagrammatic representation of the complex interplay between risk factors and biomechanical and biochemical factors in the development of knee OA.

Source: With permission from Berteau JP (2022).[5]



Flowchart 1: Pathogenesis of osteoarthritis.

Source: With permission from Michael JW-P et al. (2010).[26]

factors such as cytokines, chemokines, and proteolytic enzymes which disrupt the cartilage homeostasis, hinder attempts at repair and lead to degradation of the cartilage. The damaged cartilage is unable to fully absorb the various forces to which the joint is exposed. The body tries to compensate for the cartilage loss through an active repair process that involves thickening and hardening (sclerosis) of subchondral bone, formation of subchondral cysts and periarticular osteophytes. Loss of articular cartilage and changes in subchondral bone result in joint space narrowing, which if asymmetrical, can lead to varus or valgus deformity. Cartilage debris within the joint results in inflammation of the synovium and synovial effusions, which in turn lead to further cartilage degradation, setting up a vicious cycle that eventually leads to progressive joint destruction. Fibrosis of the joint capsule results in joint stiffness and loss of range of motion. Pain, limitation of range of motion and reduction in mobility in turn leads to disuse atrophy of muscles.

Some studies indicate that the disease may start in the subchondral bone.[28] Changes in the gene expression of the subchondral bone result in hardening and stiffening of the subchondral bone reducing its ability to withstand loads.[29] This exposes the cartilage to abnormal loads and sets up a vicious cycle of progressive degeneration. A number of studies have demonstrated a direct association between joint inflammation and disease progression.[30]

Natural History of Untreated Osteoarthritis of the Knees

There exists a significant discordance between pain, the predominant clinical symptom of knee OA, and radiological evidence of arthritic changes

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such as joint space narrowing, periarticular osteophytes, and subchondral sclerosis. Sometimes, pain may predate X-ray changes of OA. This could be due to synovial inflammation or effusion, bursitis, meniscal or ligament tears and damage to articular cartilage which may not be detected on plain radiographs. However, the more common scenario is the presence and progression of radiographic changes in the absence of clinical symptoms. Knee OA is a slowly progressive condition with the body getting time to accommodate for the various pathophysiological changes, so that symptoms may arise only after significant destruction of joint cartilage resulting in altered joint mechanics or when there is acute soft tissue pathology.

In 2019, Koga et al, reported a 40-year radiographic follow-up of 78 knees in 39 patients, who were an average 43.7 years at baseline.[31] They concluded that by the age of 80 years, almost all knees will show X-ray evidence of OA, however few patients progress to severe arthritis, not all are symptomatic, and < 5% require knee arthroplasty. It was difficult to differentiate knee OA from normal age-related changes. Leyland et al, reported on the radiographic data of 561 female subjects who were a median 53 years of age at the time of recruitment into the study.[32] At 15 years follow-up, the prevalence of Kellgren and Lawrence (KL) grade ≥ 2 OA on plain X-rays had increased from 13.7 to 47.8%. Subjects with a KL grade 1 OA at baseline were more likely to experience worsening by year 15 than those with KL grade 0.

The Framingham Osteoarthritis Study subjected 1,438 participants to a clinical and radiological survey from 1983 to 1985.[33] At a mean 8.1-year follow-up, in 1992–1993, 869 subjects participated in the repeat survey. The mean age at baseline was 70.8 ± 5 years. They reported that incidence of new symptomatic OA was 1.7 times higher in women than in men. Among women, approximately 2% per year developed incident radiographic disease (K-L grade ≤ 2 OA), 1% per year developed symptomatic knee OA (knee symptoms + K-L grade ≤ 2 OA), and about 4% per year experienced progressive knee OA (radiographs showing grade 2 disease at baseline who showed grade ≤ 3 disease on follow-up).

Similarly, Thorstensson studied 143 Swedish patients (mean age 45 years at baseline) with knee pain after a 12-year interval.[34] At baseline, 76 patients had no radiographic tibiofemoral OA but 49/76 had clinical OA as judged by criteria from the American College of Rheumatology. At follow-up, 86%, i.e., 65/76 patients with knee pain but without radiologic evidence of OA, developed K-L grade ≥ 1 radiographic OA. Radiographic progression was observed in 65/67 patients (97%) with OA at baseline. They concluded that majority of the subjects with chronic knee pain develop clinical and radiographic knee OA over 12 years. Knee pain is often the first sign of knee OA.

Driban et al, have proposed that one in seven individuals develop accelerated knee OA, wherein the disease progresses from no radiologic evidence of knee OA to advanced stage disease in < 4 years, necessitating early knee arthroplasty.[35] The risk factors for accelerated degeneration include older age, high BMI, altered joint alignment, contralateral knee OA, and low blood glucose. Typically, these patients develop rapid articular cartilage loss, large bone marrow lesions, synovitis-effusion, meniscal tears, slower chair-stand, and slower walking pace in the 2 years before onset of accelerated degeneration.

Davies-Tuck et al, studied magnetic resonance imagings (MRIs) of symptomatic knee OA patients with cartilage defects at baseline and after 2 years.[36] They reported that 81% defects showed significant progression, 15% remained stable and 4% showed reduction in severity. Older patients and those with a larger tibial plateau surface had a greater risk of progression.

Conclusion

Knee OA is the most common form of OA. Radiologically, its prevalence increases with age. However, not all subjects are symptomatic. Major risk factors for the development and propagation of knee OA include genetics, advancing age,

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female sex, obesity and metabolic syndrome, limb malalignment, acute joint trauma, or repetitive trauma from chronic overload of the joint.

Inflammatory and metabolic factors, in addition to mechanical factors are thought to contribute to the pathogenesis of OA. Further research is needed to better understand the complex pathophysiology of this condition, which will enable more focused treatment to slow down progress of the disease and reduce/delay the need for major surgeries.

KEY POINTS

- Knee OA is the most prevalent form of OA worldwide, contributing to significant functional disability. The incidence of new symptomatic disease peaks in the 55–64 years age group.
- Aside from genetic predisposition, advancing age, female sex, and obesity are significant risk factors.
- Mechanical factors that contribute to knee OA include limb malalignment, acute meniscal or ligament injuries, and injuries secondary to chronic overload of the joint.
- Failure of cartilage homeostasis leading to an imbalance between cartilage formation and breakdown is often the trigger for development of OA.
- Recent evidence suggests a contribution to the pathogenesis from synovial inflammation and changes within the subchondral bone.
- The natural history is one of slow progression, both radiologically and clinically in most patients.
- 10–15% individuals a rapidly form of knee OA. Risk factors being advanced age, high BMI, contralateral knee OA, limb malalignment.

References

1. 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):1545-602.
2. Nguyen US, Zhang Y, Zhu Y, Niu J, Zhang B, Felson DT. Increasing prevalence of knee pain and symptomatic knee osteoarthritis: survey and cohort data. Ann Intern Med. 2011;155(11):725-32.
3. Zhang Y, Jordan JM. Epidemiology of Osteoarthritis. Clin Geriatr Med. 2010;26:355-69.
4. Murphy L, Schwartz TA, Helmick CG, Renner JB, Tudor G, Koch G, et al. Lifetime risk of symptomatic knee osteoarthritis. Arthritis Rheum. 2008;59:1207-13.
5. Berteau JP. Knee Pain from Osteoarthritis: Pathogenesis, Risk Factors, and Recent Evidence on Physical Therapy Interventions. J Clin Med. 2022;11(12):3252.
6. Cisternas MG, Murphy L, Sacks JJ, Solomon DH, Pasta DJ, Helmick CG. Alternative Methods for Defining Osteoarthritis and the Impact on Estimating Prevalence in a US Population-Based Survey. Arthritis Care Res (Hoboken). 2016;68(5):574-80.
7. Reynard LN, Barter MJ. Osteoarthritis year in review 2019: Genetics, genomics and epigenetics. Osteoarthr Cartil. 2020;28:275-84.
8. Sandell LJ. Etiology of osteoarthritis: Genetics and synovial joint development. Nat Rev Rheumatol. 2012;8:77-89.
9. Jordan JM, Helmick CG, Renner JB, Luta G, Dragomir AD, Woodard J, et al. Prevalence of knee symptoms and radiographic and symptomatic knee osteoarthritis in African Americans and Caucasians: the Johnston County Osteoarthritis Project. J Rheumatol. 2007;34:172-18.
10. Losina E, Weinstein AM, Reichmann WM, Burbine SA, Solomon DH, Daigle ME, et al. Life time risk and age of diagnosis of symptomatic knee osteoarthritis in the US. Arthritis Care Res (Hoboken). 2013;65(5):1-11.
11. He Y, Li Z, Alexander PG, Ocasio-Nieves BD, Yocum L, Lin H, et al. Pathogenesis of Osteoarthritis: Risk Factors, Regulatory Pathways in Chondrocytes, and Experimental Models. Biology (Basel). 2020;9(8):194.

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12. Prieto-Alhambra D, Judge A, Javaid MK, Cooper C, Diez-Perez A, Arden NK. Incidence and risk factors for clinically diagnosed knee, hip and hand osteoarthritis: influences of age, gender and osteoarthritis affecting other joints. Ann Rheum Dis. 2014;73:1659-64.
13. Srikanth VK, Fryer JL, Zhai G, Winzenberg TM, Hosmer D, Jones G. A meta-analysis of sex differences prevalence, incidence and severity of osteoarthritis. Osteoarthr Cartil. 2005;13:769-81.
14. Szilagyi IA, Waarsing JH, Schiphof D, van Meurs JBJ, Bierma-Zeinstra SMA. Towards sex-specific osteoarthritis risk models: evaluation of risk factors for knee osteoarthritis in males and females. Rheumat. 2002;61(2):648-57.
15. Blagojevic M, Jinks C, Jeffery A, Jordan KP. Risk factors for onset of osteoarthritis of the knee in older adults: a systematic review and meta-analysis. Osteoarthr Cartil. 2010;18:24-33.
16. King LK, March L, Anandacoomarasamy A. Obesity and osteoarthritis. Indian J Med Res. 2013;138: 185-93.
17. Louati K, Vidal C, Berenbaum F, Sellam J. Association between diabetes mellitus and osteoarthritis: systematic literature review and meta-analysis. RMD Open. 2015;1(1):e000077.
18. Cavallo C, Boffa A, Salerno M, Merli G, Grigolo B, Filardo G. Adipose tissue-derived products may present inflammatory properties that affect chondrocytes and synoviocytes from patients with knee osteoarthritis. Int J Mol Sci. 2023;24(15):12401.
19. Felson DT, Zhang Y, Anthony JM, Naimark A, Anderson JJ. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. The Framingham Study. Ann Intern Med. 1992;116(7):535-9.
20. Slauterbeck JR, Kousa P, Clifton BC, Naud S, Tourville TW, Johnson RJ, et al. Geographic mapping of meniscus and cartilage lesions associated with anterior cruciate ligament injuries. J Bone Joint Surg Am. 2009;91:2094-2103.
21. Oiestad BE, Engebresten L, Starheim K, Risberg MA. Knee osteoarthritis after anterior cruciate ligament injury. A systematic review. Am J Sports Med. 2009;37:1434-43.
22. Roos H, Lauren M, Adalberth T, Roos EM, Jonsson K, Lohmander LS. Knee arthritis after meniscectomy. Prevalence of radiographic changes after 21 years, compared with matched controls. Arth. Rheum. 1998;41687-693.
23. Zhu J, Zhu Y, Xiao W, Hu Y, Li Y. Instability and excessive mechanical loading mediate subchondral bone changes to induce osteoarthritis. Ann Transl Med. 2020;8(6):350.
24. Primorac D, Molnar V, Rod E, Jeleč Ž, Čukelj F, Matišić V, et al. Knee Osteoarthritis: A review of pathogenesis and state-of-the-art non-operative therapeutic considerations. Genes (Basel). 2020;11(8):854.
25. Cerejo R, Dunlop DD, Cahue L, Channin D, Song J, Sharma L. The influence of alignment on the risk of knee osteoarthritis progression according to baseline stage of disease. Arth Rheum. 2002;46:2632-6.
26. Michael JW-P, Schlute-Brust KU, Eysel P. The epidemiology, etiology, diagnosis and treatment of osteoarthritis of the knee. Dtsch Arztebl Int. 2010;107(9):152-62.
27. Brandt KD, Dieppe P, Radin E. Etiopathogenesis of Osteoarthritis. Med Clin N Am. 2009;93:1-24.
28. Li G, Yin J, Gao J, Cheng TS, Pavlos NJ, Zhang C, et al. Subchondral bone in Osteoarthritis: Insight into risk factors and microstructural changes. Arthritis Res. Ther. 2013;15:223.
29. Goldring M, Goldring SR. Articular cartilage and subchondral bone in the pathogenesis of Osteoarthritis: Articular cartilage and subchondral bone. Ann NY Acad Sci. 2010;1192:230-7.
30. Millerand M, Berenbaum F, Jacques C. Danger signals and inflammaging in osteoarthritis. Clin Exp Rheumatol. 2019;37(S120):48-56.
31. Koga H, Omori G, Mochizuki T, Nishino K, Takagi S, Tomiyama Y, et al. Natural history of knee osteoarthritis for 40 years from the Matsudai knee osteoarthritis survey, Japan. Osteoarthr and Cartil. 2020;28(S432).
32. Leyland KM, Hart DJ, Javaid MK. The natural history of radiographic knee osteoarthritis. A 14-year population based cohort study. Arth Rheum. 2012;64(7):2243-51.
33. Felson DT, Zhang Y, Hannan MT, Naimark A, Weissman BN, Aliabadi P, et al. The incidence and natural history of knee osteoarthritis in the elderly, Framingham osteoarthritis study. Arth Rheum. 1995;38(10):1500-5.
34. Thorstensson CA, Andersson MLE, Jönsson H, Saxne T, Petersson IF. Natural course of knee osteoarthritis in middle-aged subjects with knee pain: 12-year follow-up using clinical and radiographic criteria. Ann Rheum Dis. 2009;68:1890-3.
35. Driban JB, Harkey MS, Barbe M, Ward RJ, MacKay JW, Davis JE, et al. Risk factors and the natural history of accelerated knee osteoarthritis: A narrative review. BMC Musculoskelet Disord. 2020;21(1):332.
36. Davies-Tuck ML, Wluka AE, Wang Y, AJ Teichtahl, G Jones, C Ding, et al. The natural history of cartilage defects in people with knee osteoarthritis. Osteoarthr Cartil. 2008;16(3):337-42.

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- The various treatment options for knee osteoarthritis ranging from nonoperative treatment and orthobiologics to knee preservation surgery and unicompartmental and total knee replacement are discussed.
- A case series is provided to highlight the appropriate treatment option for specific clinical and radiological presentations.

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