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# Osteochondritis Dissecans of the Knee

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#### **Abstract**

Osteochondritis dissecans (OCD) is a common but poorly understood source of knee pain and dysfunction. It is a condition primarily affecting the subchondral bone, with secondary effects on the articular cartilage surface. A large amount of research over the past two decades has produced many valuable insights into the condition, but further study and elucidation are still needed. The goal of this chapter will be to serve as a general overview of osteochondritis dissecans as it is understood today, including the etiology, clinical presentation, diagnosis, treatment options, outcomes, and future research aims.

Keywords: osteochondritis dissecans, knee, cartilage injury, OCD treatment, OCD outcomes

#### 1. Introduction

Osteochondritis dissecans (OCD) has become a well-recognized, but still poorly understood source of knee pain and dysfunction. It is a condition primarily affecting the subchondral bone, with secondary effects on the articular cartilage surface. A large amount of research over the last two decades has produced many valuable insights into the condition, but further study and elucidation are still needed. The goal of this chapter will be to serve as a general overview of osteochondritis dissecans as it is understood today, including the etiology, clinical presentation, diagnosis, treatment options, outcomes, and future research aims.

The term osteochondritis dissecans was first documented in the literature in 1887 by Franz Konig, who described a presumed inflammatory process leading to loose bodies in the elbow and knee joints in young, atraumatic patients [1]. This theory was ultimately disproved as histological studies began to support findings of necrosis rather than inflammation in OCD lesions [2–6]. Many other theories and descriptions of osteochondritis dissecans have subsequently been proposed, but a definitive understanding remains elusive. The current working



definition of OCD as developed by the leading collaborative research group on the topic is as follows: a focal, idiopathic alteration of subchondral bone with risk for instability and disruption of adjacent articular cartilage that result in premature osteoarthritis [7].

Traditionally, OCD had been subclassified into two groups based on the status of the distal femoral physis. Juvenile OCD occurs in those with an open distal femoral physis, whereas adult OCD is found in skeletally mature patients [8]. Previously, the etiology of OCD in skeletally immature individuals was thought to be from a fundamental disturbance in epiphyseal development. The adult form, on the other hand, was believed to be associated with more direct traumatic causation [9]. However, many experts now currently feel adult OCD is in the majority of cases not a distinct entity, but instead the natural progression of juvenile OCD missed in adolescence [10–12]. While the nomenclature is no longer as critical, the distinction between "juvenile" and "adult" OCD as based on presentation and timing of diagnosis is still important in regards to prognosis. Multiple studies have shown that juvenile OCD lesions to be more stable in appearance and to have a better prognosis than those diagnosed in adulthood [8, 10, 13–15].

# 2. Epidemiology

The presence of articular cartilage pathology is found in greater than 60% of patients undergoing knee arthroscopies, with focal chondral defects of all varieties found in 20% of these patients [16–18]. As a subset of these lesions, osteochondritis dissecans of the knee remains a relatively uncommon condition. In the pediatric population aged 6–19 years, the incidence of OCD lesions of the knee was found to be 9.5 per 100,000. There is a strong predilection for males versus females with an incidence of 15.4 and 3.3 per 100,000, respectively. Patients aged 12–19 years have an over three times risk of OCD than those aged 6–11 years. In terms of race and ethnicity, African Americans have double the risk of OCD of the knee compared to non-Hispanic whites, and at least 4 times the risk of disease as all other races and ethnicities [19].

The most common location for OCD lesions to occur is in the medial femoral condyle, which accounts for 70–85% of all lesions. The majority of these lesions occur in the posterolateral aspect of the medial femoral condyle [19]. The next most frequent location is the lateral femoral condyle, and the lesions in this location are often found to be larger and more advanced. OCD lesions are also rarely found on the patella, trochlea, and tibial plateau [11].

# 3. Etiology

Starting with Konig's inflammatory theory, numerous hypotheses regarding the true pathophysiology behind the formation and progression of OCD lesions of the knee have been proposed, but no one theory has gained uniform consensus. In histologic review of OCD lesions, necrosis of the subchondral bone is often identified but it remains unclear if the presence of the necrosis is primary or secondary to the pathogenesis of OCD [3–6, 20]. The vascularity of the subchondral bone has been described as an end arterial arcade with poor anastomoses.

Histology of necrotic bone has been shown to be consistent with vascular occlusions, and it has been proposed that insufficient arterial branching could lead to subchondral bone infarction and subsequent OCD [21, 22]. The presence of an ischemic zone in the lateral aspect of the medial femoral condyle has been questioned, although particularly in young patients who have good distal femoral blood supply [23–26].

Repetitive microtrauma has become the most accepted cause of OCD, mainly due to the rising incidence of the disorder among athletes [27]. The theory states that an initial stress reaction occurs in the subchondral bone of the knee and with further loading a true stress fracture is generated. Repetitive, progressive loading prevents the stress fracture from healing and eventually the subchondral bone becomes necrotic [2]. The fragment begins to dissect and ultimately separate from the fracture bed leading to an unstable OCD lesion. In this theory, bone necrosis is seen as secondary to trauma rather than to a primary lack of vascularity. Mechanical axis alignment has also been associated with OCD, with aberrant mechanical pressures on the condyles potentially leading to the formation of an OCD [28]. The true etiology of OCD is most likely multi-factorial and a combination of the currently proposed theories.

### 4. Clinical presentation and physical examination

The clinical presentation of OCD lesions can be quite variable and often differs depending on the stability and severity of the lesion. Stable lesions, as are frequently seen in juvenile OCD, often present with complaints of nonspecific and poorly localized knee pain which is exacerbated by exercise, particularly when climbing stairs or hills [10]. Unstable lesions are commonly seen in adult OCD and present with more mechanical symptoms like swelling, stiffness, locking, and catching.

On physical examination, both stable and unstable lesions may present with an antalgic gait. An external rotation of the tibia during gait can be seen as compensation for impingement of the tibial eminence on an OCD lesion of the medial femoral condyle [29]. This can be tested clinically with the Wilson test, which elicits pain when the tibia is internally rotated during extension of the knee between 90 and 30°. Pain is relieved with tibial external rotation as it moves the eminence away from the lesion. Ligamentous stability and overall alignment must also be assessed to allow for concomitant pathology to be appropriately addressed. Muscle strength testing is also important as significant dynamic strength deficits of the quadriceps and core may warrant rehabilitation attempts prior to surgery [30].

# 5. Imaging

Plain radiographs and magnetic resonance imaging (MRI) are the two most commonly used imaging modalities in evaluating knee OCD. Radiographs are commonly used for the initial diagnosis and assessment of skeletal maturity, whereas MRI highlights changes in the articular cartilage and subchondral bone.

Radiographs are relatively inexpensive and easy to obtain, making it the initial imaging choice for evaluation of suspected OCD. Radiographs evaluating for OCD lesions often include anteroposterior, lateral, sunrise, and tunnel/notch views. The characteristic appearance of an OCD lesion of the knee consists of a well-circumscribed lucent defect in the subchondral bone [31]. The notch view, which is obtained with a posterior to anterior beam at approximately 30° of flexion, is particularly helpful for evaluating the posterior aspects of the femoral condyles [8]. Evaluating for potential lesions in boys younger than 13 and girls younger than 11 requires caution as they may develop secondary ossifications that can resemble OCD lesions and MRI is often needed for clarification [10]. Given the limitations of radiographs in assessing an OCD lesion, MRI is often used to evaluate the true size and stability in order to determine an appropriate surgical plan.

### 6. Classification systems

The most commonly used classification system for OCD lesions is based on MRI findings. The Hefti system divides lesions into five different stages and differentiates between stable (stages 1 and 2) and unstable (stages 3, 4, and 5) lesions with progressive pathology noted [13].

The MRI classification has been shown to be accurate to divide lesions into stable and unstable categories, but ultimately arthroscopic evaluation provides the best assessment of the OCD lesion [32]. Multiple arthroscopic systems have been proposed to classify lesions during surgery, but no comprehensive system to describe the full complement of OCD lesions has been accepted [32–37]. The Research in OsteoChondritis of the Knee (ROCK) group developed a novel classification system to provide a common language in describing these lesions [38]. To optimize comprehensibility and applicability, each type was described with a memorable name. The classification divides lesions into immobile and mobile lesions. The "cue ball" (no detectable abnormality), "shadow" (cartilage intact but subtly demarcated), and "wrinkle in the rug" (cartilage is demarcated with a fissure or wrinkle) are in the immobile category. The mobile lesions consist of the "locked door" (cartilage fissuring at periphery but unable to hinge open), "trap door" (able to hinge open the fissure), and "crater" (exposed subchondral bone defect). This classification system has been shown to have very good inter-observer reliability and should be used to facilitate a common language which is crucial for future collaborative research.

# 7. Nonoperative treatment

Nonoperative management is the appropriate first line of treatment for stable juvenile OCD lesions. Juvenile OCD lesions have a higher healing potential than adult lesions, and an open distal femoral physis has been shown as one of the best predictors for successful nonoperative management [39]. Conservative management is usually attempted for a minimum of 3 months to allow for potential healing. Most current nonoperative treatment plans focus on activity modification with cessation of impact activities and protected weight bearing with crutches or

an offloader brace [40]. The goal of conservative management is to eliminate pain and repetitive loading to help promote healing of OCD lesions.

Overall, successful healing rates >50% have been shown for stable juvenile OCD lesions treated nonoperatively. However, this has not been replicated in the adult population, with poor results seen without surgical intervention for adult OCD lesions [4, 8, 41]. Adult OCD lesions have little capacity for healing with nonoperative means, but an unloader brace is a potential temporary option to allow an athlete to finish their season prior to operative intervention [40]. Complete resolution of symptoms takes time, patience, and compliance, which is important to stress to patients early in the process.

# 8. Operative treatment

In those patients who have failed nonoperative treatment or have large, unstable, or unsalvageable lesions, surgical intervention is often required. Cartilage treatment strategies can be characterized as palliation (debridement), repair [drilling and microfracture (MF)], or restoration [osteochondral autograft transfer (OAT), osteochondral allograft (OCA), and autologous chondrocyte implantation (ACI)] [42]. One of the most important determinations to be made prior to surgical intervention is the stability of the OCD lesion. The stability relates to the mechanical integrity of the subchondral lesion [43]. A lesion which is immobile and resting in situ is considered to be stable, whereas a lesion which is mobile, fragmented, or ex situ is considered unstable. The distinction is important for determining the appropriate surgical plan.

# 9. Subchondral drilling

Subchondral drilling is the initial standard of care operative procedure for stable OCD lesions. There are two main types of drilling, transarticular and retroarticular, but the principle behind each technique is the same. The goal of subchondral drilling is to use a Kirschner wire to disrupt the sclerotic margin of the lesion to establish channels between the necrotic subchondral bone and the healthy cancellous bone in order to promote revascularization, osseous bridging, and healing [11]. The average time to healing is around 4–6 months after surgery.

Transarticular drilling is done from inside the joint and penetrates the articular cartilage through at least one site to create subchondral penetrations. The main concern with this technique is related to the uncertain long-term implications of disrupting the articular cartilage with the drill sites. Retroarticular drilling avoids this concern by sparing the articular surface and physes with drilling through the affected femoral condyle into the lesion under fluoroscopy. Aside from the added radiation risk, this technique is also more technically demanding and risks incomplete lesion drilling, lesion displacement, or inadvertent soft tissue injury [10].

Neither technique has clearly demonstrated superior patient-orientated outcomes or radiographic healing. Transarticular drilling demonstrated an average healing rate of 91% with a mean healing time of 4.5 months with retroarticular just behind at 86% at 5.6 months [44]. No complications were noted throughout a review of all studies on drilling, making the technique not only effective but also safe option. Poorer results have been noted in older patients with closed physes, fissures of the articular cartilage, and lesions located outside the traditional posterolateral medial condyle [45–47].

#### 10. Debridement

The simplest solution to the management of an unstable OCD lesion is excision of the fragment with debridement of the remaining chondral defect. As the painful and limiting mechanical symptoms of an unstable OCD are due to these loose fragments, excision has been correlated with good short-term clinical results [48, 49]. However, as excision and debridement alone leads to a loss of articular cartilage with subsequent degenerative changes, the longer term imaging and knee function scores deteriorate [50, 51]. Even while patients maintain good clinical knee scores, evidence of early degenerative changes can be seen on radiographs at midterm follow up after excision and debridement [52, 53]. The results of these studies further reinforce that every attempt should be made to preserve, repair, or replace the native bone and cartilage that is damaged in an OCD lesion.

#### 11. Lesion fixation

For unstable lesions or stable lesions that have failed a drilling procedure, the next surgical option is often fixation of the osteochondral lesion. The general principles of lesion fixation are to attempt to restore the articular surface, enhance the blood supply of the osseous interface, and initiate early range of motion postoperatively [8].

Historically, after lesions were debrided and bone grafted, they were pinned in place with Kirschner wires; after, the lesion had been debrided and bone grafting had been applied [54]. However, this technique has largely been abandoned due to K-wire bending and inability to hold and provide an adequate compressive force to the lesion. K-wires were replaced by the use of rigid metal screw fixation, either with variable pitch or cannulated partially threaded screws. Most recently, bioabsorbable implants designed as screws or pins have become popularized for fixation. Fixation is again particularly important given the poor results seen with detached fragment removal, especially in weight-bearing areas of the femoral condyles [49, 51, 55].

Variable pitch headless screws were initially described for use in scaphoid fixation, but indications spread to include fixation of OCD lesions [56–58]. The goal of fixation with these screws is to achieve compression encouraging bony union of the subchondral fractures. The main advantage of variable pitch headless screws (Herbert screws) lies in their ability to provide strong compression and be sunk completely under the articular surface to prevent protrusion. The rigid fixation also allows early joint motion due to anatomic restoration of the joint surface [59]. The majority of patients undergoing this technique report good to excellent results without major complications [60–62]. The use of cannulated screws has also been described

with successful results; however, the major drawback is the concern for increased articular cartilage morbidity due to screw prominence on the articular surface [63, 64]. Cannulated headless compression screws have now been developed as an alternative, which theoretically combines the advantages of both techniques [65].

Bioabsorbable screws, pins, tacks, and darts have been designed and utilized with overall good results [66-69]. The main advantages of bioabsorbable fixation are the lack of metal artifact on postoperative MRI as well as theoretically no subsequent surgery needed for implant removal [66]. Bioabsorbable implants can fail though due to screw breakage, screw back out, reactive synovitis, and loss of compressive force over time [70-73]. These implant failures often lead to refractory mechanical symptoms and need for revision surgery. Despite these potential risks, unstable lesions should still be fixed instead of excised when technically feasible. As there has been no significant difference noted in comparison of bioabsorbable pins and tacks, variable pitch screws, and partially threaded screws with regard to clinical and radiographic healing, the choice of fixation is surgeon dependent [69]. The most frequently used techniques among surgeons are bioabsorbable screws and metal headless variable pitch screws [74].

#### 12. Microfracture

Microfracture is a marrow stimulation technique that was developed and implemented in the early 1980s to allow for cartilage repair. The goal of the procedure is to create microfractures in the subchondral bone perpendicular to the surface to create a surface rough enough to hold the generated marrow clot. The pluripotent cells of the clot proliferate and differentiate into cells with morphological features similar to chondrocytes. These cells then produce a cartilaginous repair tissue to fill the chondral defect [75]. The fibrocartilage which matures though is often predominately type 1 collagen, a structurally different entity from hyaline cartilage [76].

Indications for microfracture include smaller partial and full-thickness cartilage defects in patients with acceptable knee alignment. The greatest improvement occurs with the treatment of acute lesions less than 4 cm in size in patients under 35 years old [77]. Younger patients have better results with microfracture as it is crucial to have adequate height of cartilage on the lesion rim to hold the clot in place, which is difficult in degenerative lesions where the cartilage is thinner [75].

Early results of microfracture are positive with clear improvement in knee function noted throughout the literature at 2 years, particularly in smaller lesions. Despite good midterm results published by the developer of the technique, the longevity and durability of microfracture have been questioned [78-80]. When compared to other cartilage procedures like OAT and ACI, the results are mixed, although no study showed superior results for microfracture [81–83]. Microfracture has been found to have a significantly higher failure rate and need for reoperation than OAT or ACI with larger lesions (>4.5 cm<sup>2</sup>) and at greater than 5 years postoperatively [84]. An even smaller size threshold (<2 cm²) has been shown for microfracture to be successful in the demanding athletic population [81, 85].

# 13. Autograft transplantation

In the cases of failed fixation, lesion fragmentation, or chronically detached lesions, more advanced chondral procedures, like osteochondral autograft transplantation (OAT), are required. OAT was developed and then popularized in the 1990s [86, 87]. The procedure entails the harvesting of a cylindrical graft of healthy cartilage and subchondral bone from a less stressed area of the distal femur and implementing into an area of chondral defect. The graft is matched to the surface area of the defect and seated to restore a smooth cartilage surface in the joint [88]. A single plug of cartilage may be transferred or an alternative procedure termed mosaicplasty can be performed where multiple smaller plugs are implemented.

Osteochondral autograft transplantation is currently recommended as a viable option for osteochondral lesions measuring 1–4 cm<sup>2</sup> in a load-bearing area [89]. OAT offers the opportunity to repair cartilaginous defects by restoring hyaline cartilage anatomy [90]. Graft plugs should be taken from nonweight-bearing areas to avoid being arthrogenic [91]. OAT provides an immediate functional surface that allows a relatively quick rehabilitation and return to play, but a mismatch of cartilage thickness between the two sites can lead to abnormal stresses and poor function [92, 93].

Mosaicplasty has been shown to give reliably good short-term results [94–97]. In longer term studies evaluating patients who underwent mosaicplasty, there is a significant decrease in level of physical activity noted, particularly in patients whose activity level prior to surgery was high. This reduction in activity level is often due to apprehension and a desire to preserve the joint [91]. Older age, female sex, and more extensive initial lesions have been shown to be factors leading to poor prognoses after mosaicplasty [98]. Limb malalignment has also been shown to affect outcomes if not corrected, and thus concomitant osteotomy is recommended in these cases [99].

The primary concern with autografting comes from possible donor site morbidity. Cadaveric studies have shown load across donor sites during range of motion, but multiple studies have shown minimal to no complications associated with donor sites at midterm follow-up [81, 95, 97, 100]. Athletes report nearly double the rate of donor site pain compared to less active patients, indicating that vigorous exercise potentially increases donor site pain [99].

# 14. Allograft transplantation

Osteochondral allograft transplantation (OCA) involves the transfer of size-matched allograft cartilage and subchondral bone into large osteochondral defects of the knee [30]. OCA is primarily used in the management of large osteochondral defects and as a salvage option for those who have previously failed other cartilage repair techniques. Fresh osteochondral allograft transplantation is theoretically an attractive option because it can restore both the osseous and the chondral components caused by the OCD lesion [101].

Allograft tissue is harvested within 24 hours of donor death, ideally from a donor aged 15–40 years with grossly healthy articular cartilage [102]. Allografts are often matched by tissue

banks based on size, which is usually measured off an AP radiograph of the knee. The affected condyle is used for sizing and a match is sought based on the overall condyle size, with an acceptable match noted to be within ±2 mm. While it is preferred to have patient size, side, and condyle-specific matching, depending on the location of the lesion, it has been shown that plugs may be successfully transplanted to the other compartment (medial to lateral) or even to the other side (left vs. right). Once harvested, OCAs should be properly stored and implanted within 28 days for maintained chondrocyte viability and subsequent clinical benefit [103-108].

OCA is effective as a majority of patients are satisfied with their treatment and are able to return to sport or recreational activity [109]. The success of OCA is highest when a single articular surface is replaced, the surrounding ligaments and menisci are intact, and the alignment is normal [110]. Osteoarthritis or the presence of disease on both articular surfaces is a contraindication to OCA [111]. The number of previous ipsilateral knee surgical procedures, elevated BMI, age >30 years old, and medial femoral graft location have been found to be independent factors predictive of reoperation and failure after allograft transplantation [101, 112].

Overall, there is a 1 in 3 chance of undergoing an additional operation, with vast majority being arthroscopic debridement, within the first 5 years following OCA. Despite this high rate of requiring a second surgery, OCA remains an attractive option due to allograft having the ability to treat larger defects, the lack of donor site morbidity, reduced surgical time, and the ability to customize the graft to the recipient's defect site.

# 15. Autologous chondrocyte implantation

Autologous chondrocyte implantation is a two-stage procedure indicated for full thickness cartilage or OCD lesions of the knee. The initial procedure involves arthroscopic evaluation and cartilage harvesting. After 2 weeks of culturing, the harvested chondrocytes are then implanted and sealed into the cartilage defect in an attempt to recreate a hyaline cartilage interface. ACI is indicated for full thickness cartilage or osteochondral lesions of the knee ranging from 2 to 16 cm<sup>2</sup> with minimal cartilage damage on the opposing articular surface [113].

The treatment of OCD lesions with ACI has been associated with clinical improvements, including reduced pain and improved function, in both adolescents and adults at midterm follow-up [114-117]. As with other cartilage repair techniques, younger patients with more localized lesions tend to do better [118–120].

A drawback to ACI is the requirement of two separate procedures. However, most patients undergoing ACI have already failed numerous other options and are willing to undergo the extra surgery for a chance at salvage. Most complications of ACI seem to be related to the periosteal flap, including overgrowth, delamination, and arthrofibrosis. Majority of failures occur with the first 2 years after surgery [121]. Despite these limitations, ACI remains a cartilage salvage option, particularly in those who have failed other surgical modalities.

#### 16. Future research

Despite over 100 years of research, there is still much to be learned regarding osteochondritis dissecans. In 2011, the American Academy of Orthopedic Surgeons released Clinical Practice guidelines regarding OCD of the knee [122]. These guidelines found limited evidence for all aspects of the treatment of knee OCD. To provide better insight and advance the understanding of this condition, multicenter study research groups have been formed. These groups are undertaking clinical trials attempting to answer many of the unsolved issues relating to knee OCD [123].

#### 17. Conclusion

Osteochondritis dissecans of the knee remains a poorly understood and difficult problem-facing patients and orthopedic surgeons today. Affecting both articular cartilage and subchondral bone, OCD is a progressive condition leading to knee pain, mechanical symptoms, and ultimately osteoarthritis if left untreated. OCD recognized in patients with open distal femoral physes is termed juvenile OCD and has a better prognosis, particularly with nonoperative management. Adult OCD is found in patients after skeletal maturity and almost always requires surgical intervention. The stability and size of the lesion is critical in determining the appropriate surgical modality. Reparative procedures such as drilling, microfracture, and lesion stabilization have shown good early results for smaller lesions, but larger and more chronic lesions often require regenerative chondral techniques like osteochondral autograft, allograft, or acellular chondrocyte implantation. Further research is underway comparing the different techniques to determine the gold standard for each size and type of lesion. The interest and understanding of knee OCD has progressed considerably in the past 20 years, but still more prospective research studies are needed to improve the assessment and treatment of this complex condition.

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#### References

- [1] König F. The classic: On loose bodies in the joint. Clinical Orthopaedics and Related Research®. 2013;**471**(4):1107-1115
- [2] Shea KG, Jacobs JC, Carey JL, Anderson AF, Oxford JT. Osteochondritis dissecans knee histology studies have variable findings and theories of etiology. Clinical Orthopaedics and Related Research<sup>®</sup>. 2013;471(4):1127-1136

- [3] Campbell CJ, Ranawat CS. Osteochondritis dissecans: The question of etiology. Journal of Trauma and Acute Care Surgery. 1966;6(2):201-221
- [4] Linden B, Telhag H. Osteochondritis dissecans: A histologic and autoradiographic study in man. Acta Orthopaedica Scandinavica. 1977;48(6):682-686
- [5] Portigliatti Barbos M, Brach del Prever E, Borroni L, Salvadori L, Battiston B. Osteochondritis dissecans of the femoral condyles. A histological study with pre-operative fluorescent bone labelling and microradiography. Italian Journal of Orthopaedics and Traumatology. 1985;11(2):207-213
- [6] Uozumi H, Sugita T, Aizawa T, Takahashi A, Ohnuma M, Itoi E. Histologic findings and possible causes of osteochondritis dissecans of the knee. American Journal of Sports Medicine. 2009;37(10):2003-2008
- [7] Edmonds EW, Shea KG. Osteochondritis dissecans: Editorial comment. Clinical Orthopaedics and Related Research. 2013;471(4):1105
- [8] Cahill BR. Osteochondritis dissecans of the knee: Treatment of juvenile and adult forms. Journal of the American Academy of Orthopaedic Surgeons. 1995;3(4):237-247
- [9] Smillie IS. Osteochondritis Dissecans Loose Bodies in Joints. London: E. & S. Livingstone Ltd.; 1960
- [10] Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: Current concepts review. American Journal of Sports Medicine. 2006;34(7):1181-1191
- [11] Heyworth BE, Kocher MS. Osteochondritis dissecans of the knee. JBJS Reviews. 2015;3(7):e1
- [12] Edmonds EW, Polousky J. A review of knowledge in osteochondritis dissecans: 123 years of minimal evolution from König to the ROCK study group. Clinical Orthopaedics and Related Research®. 2013;471(4):1118-1126
- [13] Hefti F, Beguiristain J, Krauspe R, et al. Osteochondritis dissecans: A multicenter study of the European pediatric orthopedic society. Journal of Pediatric Orthopaedics B. 1999;8(4):231-245
- [14] Cepero S, Ullot R, Sastre S. Osteochondritis of the femoral condyles in children and adolescents: Our experience over the last 28 years. Journal of Pediatric Orthopaedics B. 2005;14(1):24-29
- [15] Bradley J, Dandy DJ. Osteochondritis dissecans and other lesions of the femoral condyles. Journal of Bone and Joint Surgery. British. 1989;71(3):518-522
- [16] Aroen A, Loken S, Heir S, et al. Articular cartilage lesions in 993 consecutive knee arthroscopies. American Journal of Sports Medicine. 2004;32(1):211-215
- [17] Widuchowski W, Widuchowski J, Trzaska T. Articular cartilage defects: Study of 25,124 knee arthroscopies. The Knee. 2007;**14**(3):177-182

- [18] Hjelle K, Solheim E, Strand T, Muri R, Brittberg M. Articular cartilage defects in 1,000 knee arthroscopies. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2002;18(7):730-734
- [19] Kessler JI, Nikizad H, Shea KG, Jacobs Jr JC, Bebchuk JD, Weiss JM. The demographics and epidemiology of osteochondritis dissecans of the knee in children and adolescents.

  American Journal of Sports Medicine. 2014;42(2):320-326
- [20] Yonetani Y, Matsuo T, Nakamura N, et al. Fixation of detached osteochondritis dissecans lesions with bioabsorbable pins: Clinical and histologic evaluation. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2010;26(6):782-789
- [21] Schenck RC Jr., Goodnight JM. Osteochondritis dissecans. Journal of Bone and Joint Surgery. American. 1996;78(3):439-456
- [22] Enneking WF. Clinical Musculoskeletal Pathology. University of Florida Press/J. Hillis Miller Health Science Center; Gainseville, Florida. 1990
- [23] Koch S, Kampen W, Laprell H. Cartilage and bone morphology in osteochondritis dissecans. Knee Surgery, Sports Traumatology, Arthroscopy. 1997;5(1):42-45
- [24] Reddy AS, Frederick RW. Evaluation of the intraosseous and extraosseous blood supply to the distal femoral condyles. American Journal of Sports Medicine. 1998;26(3):415-419
- [25] Rogers WM, Gladstone H. Vascular foramina and arterial supply of the distal end of the femur. Journal of Bone and Joint Surgery. America. 1950;32 A(4):867-874
- [26] Wall E, Von Stein D. Juvenile osteochondritis dissecans. Orthopedic Clinics of North America. 2003;34(3):341-353
- [27] Gornitzky AL, Mistovich RJ, Atuahuene B, Storey EP, Ganley TJ. Osteochondritis dissecans lesions in family members: Does a positive family history impact phenotypic potency? Clinical Orthopaedics and Related Research®. June 2017:475(6);1573-1580
- [28] Hughston JC, Hergenroeder PT, Courtenay BG. Osteochondritis dissecans of the femoral condyles. Journal of Bone and Joint Surgery. America. 1984;66(9):1340-1348
- [29] Wilson J. A diagnostic sign in osteochondritis dissecans of the knee. JBJS. 1967;49(3):477-480
- [30] Sherman SL, Garrity J, Bauer K, Cook J, Stannard J, Bugbee W. Fresh osteochondral allograft transplantation for the knee: Current concepts. Journal of the American Academy of Orthopaedic Surgeons. 2014;22(2):121-133
- [31] Zbojniewicz AM, Laor T. Imaging of osteochondritis dissecans. Clinics in Sports Medicine. 2014;33(2):221-250
- [32] O'Connor MA, Palaniappan M, Khan N, Bruce CE. Osteochondritis dissecans of the knee in children. A comparison of MRI and arthroscopic findings. Journal of Bone and Joint Surgery. British. 2002;84(2):258-262
- [33] Chen C, Liu Y, Chou P, Hsieh C, Wang C. MR grading system of osteochondritis dissecans lesions: Comparison with arthroscopy. European Journal of Radiology. 2013;82(3):518-525

- [34] Nelson DW, DiPaola J, Colville M, Schmidgall J. Osteochondritis dissecans of the talus and knee: Prospective comparison of MR and arthroscopic classifications. Journal of Computer Assisted Tomography. 1990;14(5):804-808
- [35] Jacobs JC, Archibald-Seiffer N, Grimm NL, Carey JL, Shea KG. A review of arthroscopic classification systems for osteochondritis dissecans of the knee. Orthopedic Clinics of North America. 2015;46(1):133-139
- [36] Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. Journal of Bone and Joint Surgery. America. 2003;85-A(2):58-69
- [37] Dipaola JD, Nelson DW, Colville MR. Characterizing osteochondral lesions by magnetic resonance imaging. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1991;7(1):101-104
- [38] Carey JL, Wall EJ, Grimm NL, et al. Novel arthroscopic classification of osteochondritis dissecans of the knee: A multicenter reliability study. American Journal of Sports Medicine. 2016;44(7):1694-1698
- [39] Paletta GA Jr., Bednarz PA, Stanitski CL, Sandman GA, Stanitski DF, Kottamasu S. The prognostic value of quantitative bone scan in knee osteochondritis dissecans. A preliminary experience. American Journal of Sports Medicine. 1998;26(1):7-14
- [40] Carey JL, Grimm NL. Treatment algorithm for osteochondritis dissecans of the knee. Clinics in Sports Medicine. 2014;33(2):375-382
- [41] DellaMaggiora R, Vaishnav S, Vangsness CT. Osteochondritis dissecans of the adult knee. Operative Techniques in Sports Medicine. 2008;16(2):65-69
- [42] McNickle AG, Provencher MT, Cole BJ. Overview of existing cartilage repair technology. Sports Medicine and Arthroscopy Review. 2008;16(4):196-201
- [43] Mesgarzadeh M, Sapega AA, Bonakdarpour A, et al. Osteochondritis dissecans: Analysis of mechanical stability with radiography, scintigraphy, and MR imaging. Radiology. 1987;**165**(3):775-780
- [44] Gunton MJ, Carey JL, Shaw CR, Murnaghan ML. Drilling juvenile osteochondritis dissecans: Retro-or transarticular? Clinical Orthopaedics and Related Research®. 2013;471(4):1144-1151
- [45] Boughanem J, Riaz R, Patel RM, Sarwark JF. Functional and radiographic outcomes of juvenile osteochondritis dissecans of the knee treated with extra-articular retrograde drilling. American Journal of Sports Medicine. 2011;39(10):2212-2217
- [46] Kocher MS, Micheli LJ, Yaniv M, Zurakowski D, Ames A, Adrignolo AA. Functional and radiographic outcome of juvenile osteochondritis dissecans of the knee treated with transarticular arthroscopic drilling. American Journal of Sports Medicine. 2001;29(5):562-566
- [47] Louisia S, Beaufils P, Katabi M, Robert H. Transchondral drilling for osteochondritis dissecans of the medial condyle of the knee. Knee Surgery, Sports Traumatology, Arthroscopy. 2003;11(1):33-39

- [48] Denoncourt PM, Patel D, Dimakopoulos P. Arthroscopy update #1. treatment of osteochondrosis dissecans of the knee by arthroscopic curettage, follow-up study. Orthopedic Reviews. 1986;15(10):652-657
- [49] Aglietti P, Ciardullo A, Giron F, Ponteggia F. Results of arthroscopic excision of the fragment in the treatment of osteochondritis dissecans of the knee. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2001;17(7):741-746
- [50] Michael J, Wurth A, Eysel P, König D. Long-term results after operative treatment of osteochondritis dissecans of the knee joint—30 year results. International Orthopaedics. 2008;32(2):217-221
- [51] Anderson AF, Pagnani MJ. Osteochondritis dissecans of the femoral condyles. Long-term results of excision of the fragment. American Journal of Sports Medicine. 1997;25(6): 830-834
- [52] Wright RW, McLean M, Matava MJ, Shively RA. Osteochondritis dissecans of the knee: Long-term results of excision of the fragment. Clinical Orthopaedics and Related Research®. 2004;424:239-243
- [53] Murray J, Chitnavis J, Dixon P, et al. Osteochondritis dissecans of the knee; long-term clinical outcome following arthroscopic debridement. The Knee. 2007;**14**(2):94-98
- [54] Anderson AF, Lipscomb AB, Coulam C. Antegrade curettement, bone grafting and pinning of osteochondritis dissecans in the skeletally mature knee. American Journal of Sports Medicine. 1990;18(3):254-261
- [55] Sanders TL, Pareek A, Obey MR, et al. High rate of osteoarthritis after osteochondritis dissecans fragment excision compared with surgical restoration at a mean 16-year follow-up. American Journal of Sports Medicine. 2017;45(8):1799-1805
- [56] Herbert TJ, Fisher WE. Management of the fractured scaphoid using a new bone screw. Journal of Bone and Joint Surgery. British. 1984;66(1):114-123
- [57] Wombwell JH, Nunley JA. Compressive fixation of osteochondritis dissecans fragments with Herbert screws. Journal of Orthopaedic Trauma. 1987;1(1):74-77
- [58] Thomson NL. Osteochondritis dissecans and osteochondral fragments managed by Herbert compression screw fixation. Clinical Orthopaedics and Related Research®. 1987;224:71-78
- [59] Kouzelis A, Plessas S, Papadopoulos AX, Gliatis I, Lambiris E. Herbert screw fixation and reverse guided drillings, for treatment of types III and IV osteochondritis dissecans. Knee Surgery, Sports Traumatology, Arthroscopy. 2006;14(1):70-75
- [60] Zuniga JR, Sagastibelza J, Blasco JL, Grande MM. Arthroscopic use of the Herbert screw in osteochondritis dissecans of the knee. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1993;9(6):668-670
- [61] Mackie IG, Pemberton DJ, Maheson M. Arthroscopic use of the Herbert screw in osteo-chondritis dissecans. Journal of Bone and Joint Surgery. British. 1990;72(6):1076

- [62] Makino A, Muscolo DL, Puigdevall M, Costa-Paz M, Ayerza M. Arthroscopic fixation of osteochondritis dissecans of the knee: Clinical, magnetic resonance imaging, and arthroscopic follow-up. American Journal of Sports Medicine. 2005;33(10):1499-1504
- [63] Cugat R, Garcia M, Cusco X, et al. Osteochondritis dissecans: A historical review and its treatment with cannulated screws. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1993;9(6):675-684
- [64] Johnson LL, Uitvlugt G, Austin MD, Detrisac DA, Johnson C. Osteochondritis dissecans of the knee: Arthroscopic compression screw fixation. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1990;6(3):179-189
- [65] Barrett I, King AH, Riester S, et al. Internal fixation of unstable osteochondritis dissecans in the skeletally mature knee with metal screws. Cartilage. 2016;7(2):157-162
- [66] Tabaddor RR, Banffy MB, Andersen JS, et al. Fixation of juvenile osteochondritis dissecans lesions of the knee using poly 96L/4D-lactide copolymer bioabsorbable implants. Journal of Pediatric Orthopaedics. 2010;30(1):14-20
- [67] Camathias C, Gögüs U, Hirschmann MT, et al. Implant failure after biodegradable screw fixation in osteochondritis dissecans of the knee in skeletally immature patients. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2015;31(3):410-415
- [68] Wouters DB, van Horn JR, Bos RR. The use of biodegradables in the treatment of osteochondritis dissecans of the knee: Fiction or future? Acta Orthopaedica Belgica. 2003;69(2):175-181
- [69] Kocher MS, Czarnecki JJ, Andersen JS, Micheli LJ. Internal fixation of juvenile osteochondritis dissecans lesions of the knee. American Journal of Sports Medicine. 2007;35(5):712-718
- [70] Friederichs MG, Greis PE, Burks RT. Pitfalls associated with fixation of osteochondritis dissecans fragments using bioabsorbable screws. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2001;17(5):542-545
- [71] Scioscia TN, Giffin JR, Allen CR, Harner CD. Potential complication of bioabsorbable screw fixation for osteochondritis dissecans of the knee. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2001;17(2):1-5
- [72] Fridén T, Rydholm U. Severe aseptic synovitis of the knee after biodegradable internal fixation: A case report. Acta Orthopaedica Scandinavica. 1992;63(1):94-97
- [73] Barfod G, Svendsen RN. Synovitis of the knee after intraarticular fracture fixation with biofix®: Report of two cases. Acta Orthopaedica Scandinavica. 1992;63(6):680-681
- [74] Yellin JL, Gans I, Carey JL, Shea KG, Ganley TJ. The surgical management of osteochondritis dissecans of the knee in the skeletally immature: A survey of the pediatric orthopaedic society of North America (POSNA) membership. Journal of Pediatric Orthopaedics. 2015

- [75] Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: Surgical technique and rehabilitation to treat chondral defects. Clinical Orthopaedics and Related Research®. 2001;391:S362-S369
- [76] Frisbie DD, Oxford JT, Southwood L, et al. Early events in cartilage repair after subchondral bone microfracture. Clinical Orthopaedics and Related Research®. 2003;407:215-227
- [77] Steadman JR, Rodkey WG, Singleton SB, Briggs KK. Microfracture technique for full-thickness chondral defects: Technique and clinical results. Operative Techniques in Orthopaedics. 1997;7(4):300-304
- [78] Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: Average 11-year follow-up. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2003;19(5):477-484
- [79] Goyal D, Keyhani S, Lee EH, Hui JHP. Evidence-based status of microfracture technique: A systematic review of level I and II studies. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2013;**29**(9):1579-1588
- [80] Mithoefer K, McAdams T, Williams RJ, Kreuz PC, Mandelbaum BR. Clinical efficacy of the microfracture technique for articular cartilage repair in the knee: An evidence-based systematic analysis. American Journal of Sports Medicine. 2009;37(10):2053-2063
- [81] Gudas R, Kalesinskas RJ, Kimtys V, et al. A prospective randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of osteochondral defects in the knee joint in young athletes. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2005;21(9):1066-1075
- [82] Gudas R, Gudaitė A, Pocius A, et al. Ten-year follow-up of a prospective, randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of osteochondral defects in the knee joint of athletes. American Journal of Sports Medicine. 2012;40(11):2499-2508
- [83] Ulstein S, Årøen A, Røtterud JH, Løken S, Engebretsen L, Heir S. Microfracture technique versus osteochondral autologous transplantation mosaicplasty in patients with articular chondral lesions of the knee: A prospective randomized trial with long-term follow-up. Knee Surgery, Sports Traumatology, Arthroscopy. 2014;22(6):1207-1215
- [84] Devitt BM, Bell SW, Webster KE, Feller JA, Whitehead TS. Surgical treatments of cartilage defects of the knee: Systematic review of randomised controlled trials. The Knee. 2017;3:508-517
- [85] Mithoefer K, Williams RJ 3rd, Warren RF, Wickiewicz TL, Marx RG. High-impact athletics after knee articular cartilage repair: A prospective evaluation of the microfracture technique. American Journal of Sports Medicine. 2006;34(9):1413-1418
- [86] Matsusue Y, Yamamuro T, Hama H. Arthroscopic multiple osteochondral transplantation to the chondral defect in the knee associated with anterior cruciate ligament disruption. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1993;**9**(3):318-321

- [87] Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: Ten years of experimental and clinical experience. Journal of Bone and Joint Surgery. America. 2003;85-A(2):25-32
- [88] Richter DL, Tanksley JA, Miller MD. Osteochondral autograft transplantation: A review of the surgical technique and outcomes. Sports Medicine and Arthroscopy Review. 2016;24(2):74-78
- [89] Versier G, Dubrana F. Treatment of knee cartilage defect in 2010. Orthopaedics & Traumatology: Surgery & Research. 2011;97(8):S140-S153
- [90] Baltzer A, Ostapczuk M, Terheiden H, Merk H. Good short-to medium-term results after osteochondral autograft transplantation (OAT) in middle-aged patients with focal, non-traumatic osteochondral lesions of the knee. Orthopaedics & Traumatology: Surgery & Research. 2016;102(7):879-884
- [91] Cognault J, Seurat O, Chaussard C, Ionescu S, Saragaglia D. Return to sports after autogenous osteochondral mosaicplasty of the femoral condyles: 25 cases at a mean follow-up of 9 years. Orthopaedics & Traumatology: Surgery & Research. 2015;101(3):313-317
- [92] Pareek A, Reardon PJ, Maak TG, Levy BA, Stuart MJ, Krych AJ. Long-term outcomes after osteochondral autograft transfer: A systematic review at mean follow-up of 10.2 years. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2016;32(6):1174-1184
- [93] Thaunat M, Couchon S, Lunn J, Charrois O, Fallet L, Beaufils P. Cartilage thickness matching of selected donor and recipient sites for osteochondral autografting of the medial femoral condyle. Knee Surgery, Sports Traumatology, Arthroscopy. 2007;15(4):381-386
- [94] Hangody L, Kish G, Kárpáti Z, Udvarhelyi I, Szigeti I, Bély M. Mosaicplasty for the treatment of articular cartilage defects: Application in clinical practice. Orthopedics. 1998;21(7):751-756
- [95] Marcacci M, Kon E, Zaffagnini S, et al. Multiple osteochondral arthroscopic grafting (mosaicplasty) for cartilage defects of the knee: Prospective study results at 2-year follow-up. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2005;21(4):462-470
- [96] Berlet GC, Mascia A, Miniaci A. Treatment of unstable osteochondritis dissecans lesions of the knee using autogenous osteochondral grafts (mosaicplasty). Arthroscopy: The Journal of Arthroscopic & Related Surgery. 1999;15(3):312-316
- [97] Miniaci A, Tytherleigh-Strong G. Fixation of unstable osteochondritis dissecans lesions of the knee using arthroscopic autogenous osteochondral grafting (mosaicplasty). Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2007;23(8):845-851
- [98] Solheim E, Hegna J, Øyen J, Austgulen OK, Harlem T, Strand T. Osteochondral autografting (mosaicplasty) in articular cartilage defects in the knee: Results at 5 to 9 years. The Knee. 2010;17(1):84-87
- [99] Hangody L, Dobos J, Balo E, Panics G, Hangody LR, Berkes I. Clinical experiences with autologous osteochondral mosaicplasty in an athletic population: A 17-year prospective multicenter study. American Journal of Sports Medicine. 2010;38(6):1125-1133

- [100] Kish G, Módis L, Hangody L. Osteochondral mosaicplasty for the treatment of focal chondral and osteochondral lesions of the knee and talus in the athlete: Rationale, indications, techniques, and results. Clinics in Sports Medicine. 1999;18(1):45-66
- [101] Sadr KN, Pulido PA, McCauley JC, Bugbee WD. Osteochondral allograft transplantation in patients with osteochondritis dissecans of the knee. American Journal of Sports Medicine. 2016;44(11):2870-2875
- [102] Gortz S, Bugbee WD. Allografts in articular cartilage repair. Journal of Bone and Joint Surgery. America. 2006;88(6):1374-1384
- [103] Pallante AL, Bae WC, Chen AC, Gortz S, Bugbee WD, Sah RL. Chondrocyte viability is higher after prolonged storage at 37 degrees C than at 4 degrees C for osteochondral grafts. American Journal of Sports Medicine. 2009;37(1):24S-32S
- [104] Garrity JT, Stoker AM, Sims HJ, Cook JL. Improved osteochondral allograft preservation using serum-free media at body temperature. American Journal of Sports Medicine. 2012;40(11):2542-2548
- [105] LaPrade RF, Botker J, Herzog M, Agel J. Refrigerated osteoarticular allografts to treat articular cartilage defects of the femoral condyles. A prospective outcomes study. Journal of Bone and Joint Surgery. America. 2009;91(4):805-811
- [106] Williams RJ 3rd, Dreese JC, Chen CT. Chondrocyte survival and material properties of hypothermically stored cartilage: An evaluation of tissue used for osteochondral allograft transplantation. American Journal of Sports Medicine. 2004;32(1):132-139
- [107] Ball ST, Amiel D, Williams SK, et al. The effects of storage on fresh human osteochondral allografts. Clinical Orthopaedics and Related Research®. 2004;418:246-252
- [108] Pallante AL, Chen AC, Ball ST, et al. The in vivo performance of osteochondral allografts in the goat is diminished with extended storage and decreased cartilage cellularity. American Journal of Sports Medicine. 2012;40(8):1814-1823
- [109] Nielsen ES, McCauley JC, Pulido PA, Bugbee WD. Return to sport and recreational activity after osteochondral allograft transplantation in the knee. American Journal of Sports Medicine. 2017:45(7):1608-1614
- [110] Emmerson BC, Gortz S, Jamali AA, Chung C, Amiel D, Bugbee WD. Fresh osteochon-dral allografting in the treatment of osteochondritis dissecans of the femoral condyle. American Journal of Sports Medicine. 2007;35(6):907-914
- [111] Garrett JC. Fresh osteochondral allografts for treatment of articular defects in osteochondritis dissecans of the lateral femoral condyle in adults. Clinical Orthopaedics and Related Research<sup>®</sup>. 1994;**303**:33-37
- [112] Frank RM, Lee S, Levy D, et al. Osteochondral allograft transplantation of the knee: Analysis of failures at 5 years. American Journal of Sports Medicine. Mar 2016;45(4): 864-874

- [113] Peterson L. Technique of autologous chondrocyte transplantation. Techniques in Knee Surgery. 2002;1(1):2-12
- [114] Mithöfer K, Minas T, Peterson L, Yeon H, Micheli LJ. Functional outcome of knee articular cartilage repair in adolescent athletes. American Journal of Sports Medicine. 2005;33(8):1147-1153
- [115] Cole BJ, DeBerardino T, Brewster R, et al. Outcomes of autologous chondrocyte implantation in study of the treatment of articular repair (STAR) patients with osteochondritis dissecans. American Journal of Sports Medicine. 2012;40(9):2015-2022
- [116] Peterson L, Brittberg M, Kiviranta I, Åkerlund EL, Lindahl A. Autologous chondrocyte transplantation biomechanics and long-term durability. American Journal of Sports Medicine. 2002;30(1):2-12
- [117] Peterson L, Minas T, Brittberg M, Lindahl A. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation. Journal of Bone and Joint Surgery. America. 2003;85(suppl 2):17-24
- [118] Behery OA, Harris JD, Karnes JM, Siston RA, Flanigan DC. Factors influencing the outcome of autologous chondrocyte implantation: A systematic review. Journal of Knee Surgery. 2013;26(03):203-212
- [119] Harris JD, Siston RA, Pan X, Flanigan DC. Autologous chondrocyte implantation: A systematic review. Journal of Bone and Joint Surgery. America. 2010;**92**(12):2220-2233
- [120] Krishnan SP, Skinner JA, Bartlett W, et al. Who is the ideal candidate for autologous chondrocyte implantation? Journal of Bone and Joint Surgery. British. 2006;88(1):61-64
- [121] Polousky JD, Albright J. Salvage techniques in osteochondritis dissecans. Clinics in Sports Medicine. 2014;33(2):321-333
- [122] Chambers HG, Shea KG, Carey JL. AAOS clinical practice guideline: Diagnosis and treatment of osteochondritis dissecans. Journal of the American Academy of Orthopaedic Surgeons. 2011;19(5):307-309
- [123] Nepple JJ, Milewski MD, Shea KG. Research in osteochondritis dissecans of the knee: 2016 update. The Journal of Knee Surgery. 2016;**29**(07):533-538

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