

Juvenile Osteochondritis Dissecans: Current Concepts

Ibrahim Akkawi¹, Hassan Zmerly², Maurizio Draghetti¹, Lamberto Felli³

1. Orthopedics, Casa di Cura Villa Erbosa, Bologna, ITA 2. Orthopedics, Villa Erbosa Hospital, Bologna, ITA 3. Orthopedics, San Martino Hospital, Genova, ITA

Corresponding author: Ibrahim Akkawi, i.akkawi@libero.it

Review began 07/16/2024

Review ended 07/22/2024

Published 07/27/2024

© Copyright 2024

Akkawi et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.65496

Abstract

Osteochondritis dissecans (OCD) primarily damages the subchondral bone, leading to damage to the articular cartilage. Juvenile OCD (JOCD) of the knee is limited to skeletally immature and young patients with open growth plates on radiographs. We conducted a review of PubMed articles up until March 16, 2024, using a combination of the following keywords: knee, juvenile, and osteochondritis dissecans. This narrative review included a total of 56 relevant articles that investigated the etiology, incidence, clinical presentation, imaging, classification, and treatment of JOCD of the knee in patients less than 20 years of age. The exact etiology is controversial. Most authors believe that the disease involves multiple theories, such as ischemia, recurrent trauma, and genetic predisposition. Radiographs, the first imaging study in this patient group, cannot determine the stability or instability of the surface cartilage of the OCD lesion. As a result, MRI has become a recommended diagnostic method for determining OCD stability and providing important information for determining a treatment plan. For stable JOCD lesions, nonsurgical treatment is often advised. For unstable and stable lesions that do not respond to nonsurgical treatment, several surgical techniques with good healing rates are available.

Categories: Pediatric Surgery, Rheumatology, Orthopedics

Keywords: autologous adipose-derived mesenchymal stem cell implantation, autologous chondrocyte implantation, autologous osteochondral transfer, microfracture, drilling, juvenile osteochondritis dissecans

Introduction And Background

Osteochondritis dissecans (OCD) is believed to predominantly impact subchondral bone, leading to further damage to the articular cartilage [1]. The most frequently affected part is the knee [2]. Juvenile OCD (JOCD) is a condition that affects skeletally immature patients and has visible growth plates on their X-rays [3]. Based on the histological cartilage study [4], 20 years of age is considered the transitional age between adult and juvenile cartilage, meaning that patients with JOCD are those who are younger than 20 years at the time of presentation. The majority of the reviews in the literature included studies that addressed JOCD of the knee in patients over 20 years of age [5] and addressed the only surgical outcome of JOCD without mentioning the outcome of conservative treatment [6]. To the best of the authors' knowledge, no review has addressed the new surgical techniques for treating JOCD of the knee, such as the use of cell-free osteochondral collagen scaffolds [7], one-step bone marrow-derived cell transplantation [8], and autologous adipose-derived mesenchymal stem cell (AD-MSCs) implantation [9]. Hence, this narrative review seeks to provide an updated report on the literature regarding the etiology, incidence, clinical presentation, imaging, classification, and conservative and surgical treatment, with a focus on new surgical techniques for knee JOCD in patients less than 20 years of age.

Review

Methods

A comprehensive search of the PubMed database was performed until March 16, 2024, utilizing a combination of the keywords "knee," "juvenile," and "osteochondritis dissecans." In order to ensure that no relevant documents were missed, a comprehensive analysis was conducted on each reference list from the selected research. The inclusion criteria consisted of studies conducted in English that focused on patients with JOCD of the knee who were under the age of 20. The exclusion criteria encompassed studies conducted in languages other than English, research focusing on individuals above the age of 20 with OCD, and studies specifically addressing JOCD in joints other than the one under investigation.

Results

Out of the total of 231 papers found in the original search, 175 were excluded since they did not match the specified inclusion criteria. This narrative review includes a comprehensive analysis of 56 relevant papers [1-56] that examine the etiology, incidence, clinical presentation, imaging, classification, and management of JOCD in individuals under the age of 20.

How to cite this article

Akkawi I, Zmerly H, Draghetti M, et al. (July 27, 2024) Juvenile Osteochondritis Dissecans: Current Concepts. Cureus 16(7): e65496. DOI 10.7759/cureus.65496

Discussion

Etiology

The precise origins of it are mostly unclear. The prevailing consensus among experts is that the condition encompasses several theories, including repeated trauma, ischemia, genetic susceptibility, and anatomical causes [10-12]. As a result of repeated trauma, the subchondral bone is believed to develop a stress fracture, leading to necrosis and detachment of the bone from the articular cartilage that surrounds it [13]. Inflammatory diseases [10], being overweight [14], having a lateral discoid meniscus [15], undergoing meniscal surgery for a juvenile discoid lateral meniscus [16], having a narrower intercondylar notch width [17], having a posterior cruciate ligament insertion further distally on the femur in patients with medial femoral condyle (MFC) OCD compared to patients with lateral femoral condyle (LFC) OCD and normal knees [18], having medial and lateral tibiofemoral subluxation [19], and having leg malalignment [20] may also cause OCD. Jacobi et al. [20] have shown a correlation between varus alignment and medial condyle lesions, as well as between valgus alignment and lateral condyle lesions. Additional morphological features in individuals with MFC OCD lesions include a higher degree of posterior and medial tibial slope [21], a more noticeable tibial eminence [22], and increased mobility of the anterior horn of the meniscus [23].

A retrospective investigation [24] revealed a surprisingly high incidence of vitamin D deficiency among individuals first diagnosed with JOCD, suggesting a potential association between vitamin D deficiency and the development of JOCD. Hussain et al. [25] found a potential connection between hGH insufficiency, hormone replacement therapy, and the development of OCD lesions.

Incidence

Based on a recent survey, knee JOCD is ranked as the fourth most common injury among young athletes [26]. The majority of patients engage in activities such as baseball, football, and basketball, which involve significant axial loading and jumping [10,26,27]. The incidence is higher among males compared to girls [10]. According to retrospective research [28], the prevalence of OCD in children between the ages of 6 and 19 is 15.4 per 100,000 for men and 3.3 per 100,000 for girls. Furthermore, the prevalence of OCD in the 12-19 age group was 3.3 times higher compared to the 6-11 age group.

In recent years, the rising participation of young children, regardless of gender, in competitive sports has influenced the prevalence of this illness. Thus, there is evidence to suggest that the average age at which JOCD becomes apparent is decreasing, and it is more common among females [29]. According to several authors, the real prevalence and incidence of a condition may be underestimated due to patients without symptoms and incorrect diagnoses made during clinical evaluations [3]. The MFC is the joint most frequently afflicted by OCD of the knee, accounting for 69% of cases. The LFC follows at 15%, the patella at 5%, and the femoral trochlea at 1% [30].

Clinical Assessment

Based on the studies conducted by D'Angelo et al. [3] and Kumar et al. [10], it is often observed that patients usually suffer from pain and stiffness during physical activity. These symptoms are aggravated by periods of severe exercise or activities, such as climbing hills or stairs. In addition, there may be sporadic instances of clicking, locking, and catching, which suggest the presence of unstable lesions or loose intraarticular bodies.

Evaluation may reveal joint effusion in 23-44% of patients and point pain on the affected femoral condyle in 40-70% of patients [2]. Only advanced or prolonged lesions show atrophy of the quadriceps muscles of the affected limb [3]. During the initial phases of the disease, the knee's ability to move is often normal. However, as the condition progresses and loose bodies emerge, pain or mechanical blockage may restrict the knee's range of motion to passive extension [3]. Stable lesions or a loose body may induce crepitus during the knee range of motion [13]. Both stable and unstable lesions can result in an antalgic gait [29]. Extending the knee while internally twisting the tibia (known as the Wilson sign) can cause pain. However, in a sample of 32 patients who had OCD lesions in the MFC, the Wilson sign was insensitive, as 75% of the tests yielded negative results [31]. Certain individuals may experience difficulty walking, such as the need to externally rotate the tibia to avoid the tibial spine from coming into contact with the lateral side of the MFC [2].

Imaging

For patients with a clinical history and physical examination results consistent with JOCD, radiographs serve as the initial imaging assessment to assess lesion healing [30]. Radiographic evaluations of patients with knee OCD generally include anteroposterior, lateral, tunnel, merchant, and sunrise views [2].

A clearly defined subchondral bone region, split by a radiolucent contour in the shape of a crescent, characterizes an OCD lesion on radiographs. Nevertheless, the presence of subchondral irregularities or lucencies in one or both femoral condyles may indicate normal growth in people who are still developing their skeletal structure. This phenomenon is generally referred to as developmental ossification variation or

irregular ossification [30]. Moreover, radiographs are unable to determine the stability or instability of the surface cartilage in an OCD lesion, a critical factor in selecting an appropriate treatment plan [5]. Frequent observations have revealed disparities between the data acquired by radiography and that gained during surgery. For instance, fragments that seem to have detached from their docking location on X-rays remain secure and protected by healthy cartilage during arthroscopic surgery [32]. As a result, MRI has become more important as the preferred diagnostic technique for assessing OCD. In addition, it tracks the progression and recovery of OCD, assists in identifying the likely site of the lesion, and assesses whether the lesion is stable or unstable [32,33].

De Smet et al. [34] first outlined the MRI criteria for assessing the instability of OCD lesions, which include the existence of a high signal intensity between the OCD lesion and nearby bone; a joint fracture indicated by the passage of high signal joint fluid through the subchondral bone plate; a localized defect in the bone and cartilage filled with joint fluid; or a cyst filled with fluid that is at least five millimeters deep within the lesion. Despite its high sensitivity (97-100%), the specificity of this MRI finding is limited (11-55%), particularly in patients who are still growing. [2]. Heywood et al. [35] found that there was a 30% agreement between the MRI and arthroscopy phases, as evaluated by Dipaola et al. [36]. Roßbach et al. [33] used arthroscopy as a standard to test how well preoperative MRI could diagnose OCD of the knee and talus in children. They employed the OCD classification system of Dipaola et al. [36] and found that the total accuracy of MRI was just 41.3%. In addition, Samora et al. [37] found that preoperative MRI had limited accuracy in identifying JOCD. There was only a 62.1% agreement between Dipaola et al.'s MRI [36] and Guhl's arthroscopic grading [38].

According to O'Connor et al. [39], unstable lesions are defined as those with a disruption in the articular cartilage on T1 and a high signal intensity on T2 located behind the fragment. This refinement significantly improved MRI's accuracy in assessing the stability of JOCD lesions, raising it from 45% to 85%. In addition, Kijowski et al. suggested a novel MRI classification approach specifically tailored for JOCD [40]. In patients with JOCD, the presence of three specific signs indicates 100% accuracy in diagnosing lesion instability. These signs include a JOCD lesion surrounded by a high T2 signal intensity border that has the same signal intensity as the nearby joint fluid, a secondary outer border with low T2 signal intensity, and multiple breaks in the subchondral bone plate. In addition, they found that perilesional cysts larger than 5 mm in diameter (with a sensitivity of 25% and a specificity of 100%) or several cysts (with a sensitivity of 38% and a specificity of 100%) were also indicators of instability.

Both patients who underwent surgery and those who received conservative therapy commonly underwent MRI scans to assess their recovery progress [33]. The MRI results, primarily visible on non-fat-suppressed T1-weighted or GRE-weighted images, reveal that the healing process following nonoperative treatment entails a decrease or elimination of the surrounding bone marrow edema, a reduction in the lesion's size, a decrease or elimination of the intense signal border or cyst-like areas, and the growth of bone within the OCD lesion, which connects to the nearby subchondral bone [30].

Classification

While plain radiography is commonly used to assess healing progress, it is not the most effective technique for determining the severity of lesions because it does not offer a comprehensive evaluation of the articular cartilage. MRI can provide a more accurate classification of JOCD lesions. The MRI classifications most commonly utilized are Hefti et al. [41] and Dipaola et al. [36]. The arthroscopic classifications most commonly utilized are Guhl's [38], the International Cartilage Repair Society's [42], and Ewing and Voto's [43].

Nonoperative Treatment

Stable JOCD lesions with intact articular cartilage frequently require nonoperative treatment [27]. Experts recommend a minimum of three to six months of nonoperative treatment [33]. Nonoperative methods such as immobilization and activity restriction often treat stable lesions in skeletally immature patients. A brace, cast, or standard knee immobilizer can achieve successful immobilization [29]. Research studies have shown that prolonged immobilization might have negative effects on the knee's functionality [2]. During a period of six to eight weeks, those undergoing nonoperative treatment should refrain from participating in sports and engaging in high-impact activities. However, for patients who provide their agreement, it is possible to resume regular weight-bearing activities [44]. However, if the patient continues to experience symptoms during routine activities such as walking, we gradually increase their weight-bearing limits until they achieve asymptomatic status [13]. During the initial three months, it is recommended to engage in other forms of exercise such as swimming, stationary cycling, deep-water running, and other activities that have less impact on the body. It is advisable to engage in regular activities and sports after a period of around four to six months [3,10].

Reportedly, the success rate of nonoperative therapy in this specific group of patients varies between 49% and 95% [1,45,46]. Krause et al. [45] found that after one year of nonoperative therapy, 49% of patients with JOCD either fully healed or were in the process of healing. Hughes et al. [46] found a 95% recovery rate over a

five-year period in 21 knees affected by JOCD. The discrepancies in results may be attributed to the many MRI instability criteria utilized by writers, each having a unique level of specificity and sensitivity.

Research has shown that the location and size of a lesion have an impact on its ability to recover. Hefti et al. [41] found that lesions with a diameter bigger than 20 mm had a worse prognosis, as shown by a significant multicenter study. Kumar et al. [10] found that lesions with smaller longitudinal diameters and surface areas, as identified by MRI, have a higher likelihood of healing without the need for surgery. Furthermore, lesions located in atypical areas have a noticeable deficiency in their ability to recover. Vannini et al. [8] discovered that the magnitude of the lesion had a detrimental impact on the extent of improvement in the clinical result. In their retrospective cohort examination of 37 patients treated surgically for knee JOCD, Bangert et al. [12] found that defects with a depth of less than 0.8 cm^2 resulted in significantly better outcomes compared to defects with a depth of 0.8 cm^2 or larger. The location, size, and BMI at the time of surgery did not have an impact on the outcome. Patients with JOCD who had a discoid meniscus and a longer duration between onset and consultation had a worse outcome when treated nonoperatively in the LFC [1].

Operative Treatment

A range of surgical techniques are used for unstable JOCD of the knee as well as for stable lesions that do not heal with nonoperative therapy, with healing success rates of 62%, according to a systematic review by Eismann et al. [5]. Trinh et al. [6] conducted a review that showed that surgical treatment for knee OCD in young patients significantly enhances clinical and radiological outcomes in the short, medium, and long term. Their analysis determined that, when comparing many surgical procedures, there were no discernible disparities in clinical or radiographic results.

Reparative procedures include transarticular and retroarticular drilling [47], reduction and fixation [48–50], and microfracture [51]. Autologous osteochondral transfer, autologous chondrocyte implantation (ACI), cell-free osteochondral collagen scaffold, one-step bone marrow-derived cell transplantation, and AD-MSCs implantation are examples of restorative treatments.

Transarticular and retroarticular drilling of the subchondral bone can be employed to treat stable lesions in individuals who have not shown improvement with nonoperative therapy. The objective is to facilitate the recovery of JOCD lesions that are less than 2.5 cm^2 [10,30]. In order to stimulate the regrowth of blood vessels and the healing of the damaged subchondral bone, vascular paths are created using the bone marrow underneath it [30]. Gunton et al. [47] conducted a comprehensive analysis of the short-term clinical outcomes of drilling stable JOCD lesions. The study found that there were no notable disparities in the rates of radiographic healing for JOCD lesions treated with retroarticular or transarticular drilling procedures. The healing rates were 86% and 91%, respectively.

Various techniques of internal fixation utilizing diverse metallic implants have been employed to treat OCD lesions [49]. Complications related to these implants include the necessity for subsequent surgery to remove them, interference with MRI scans, insufficient compression, fracture of the pins, loosening of the implants, and migration through the skin [48,56]. Due to these complexities, bioabsorbable implants have been created. Regrettably, due to their inability to compress, bioabsorbable implants have been associated with negative outcomes, such as synovitis and loss of fixation [49]. Webb et al. [48] conducted a retrospective review of 20 patients who had internal fixation of their unstable JOCD lesions using either bioabsorbable fixation, metal fixation, or a combination of both. The patients were followed up clinically for an average of seven years. During the final evaluation, 15 out of 20 knees (75%) showed osseous integration when assessed by radiographic imaging. Tabaddor et al. [49] conducted a retrospective case series to assess the outcomes of patients with unstable JOCD who were treated with bioabsorbable pins. The study included twenty-four knees, and the patients were followed up for an average of 39.6 months. The MRI scan showed that 15 out of 17 patients (88.2%) experienced either partial or complete recovery, while 22 out of 24 patients (91.7%) had favorable to outstanding clinical results. Similarly, in a study conducted by Komnos et al. [50], it was shown that 90% of patients with JOCD of the knee who had retrograde drilling and arthroscopic stabilization of the lesion with bioabsorbable pins showed evidence of lesion healing based on MRI data. Out of the 40 patients included in the study, 36 showed successful healing.

Microfracturing and OAT procedures are conducted to address defects that are less than $2\text{--}3.5\text{ cm}^2$ [12,51]. The suggested technique of microfracture entails creating several microscopic perforations in the subchondral bone with the aim of attracting stem cells and growth factors to the damaged cartilage area. Gudas et al. [51] conducted a clinical trial where they randomly assigned participants and observed them over a period of 4.2 years. They found that 63% of the 23 patients who had microfracture surgery for the treatment of JOCD anomalies in the femoral condyles of the knee joint experienced excellent or satisfactory clinical results. In the study conducted by the same authors [51], it was shown that 83% of the patients with JOCD (19 out of 23) had excellent or good clinical results after undergoing OAT. The average follow-up period was 4.2 years. Furthermore, 91% of the patients, namely 19 out of 21, exhibited good or adequate repairs as shown on MRI scans. In 11 patients, Miniaci and Tytherleigh-Strong [52] reported the results of using OAT to treat symptomatic, unstable JOCD lesions. After six months of surgery, they found that

repeated MRI scans showed complete healing of the osseous component of the lesion in all knees. Similarly, Miura et al. [53] discovered that none of the six cases of JOCD lesions treated with OAT showed contact between the osteochondral fragment and subchondral bone on MRI three months after surgery.

ACI is often the preferred method for defects larger than 3 cm² [12]. Beck et al. [4] conducted longitudinal cohort research to determine the long-term, patient-based outcomes of ACI treatment for knee JOCD in 10 patients. They discovered a failure rate of 20% over an average period of 12 years. The study conducted by Moriya et al. [54] found that six individuals with knee JOCD who underwent ACI treatment experienced similar results. The ultimate follow-up demonstrated an 85% success rate. In a study conducted by Krishnan et al. [55], it was shown that after an average follow-up period of 4.3 years, 82.1% of patients had excellent or good outcomes, and 96.4% of the 28 patients with JOCD of the knee had successful results.

In a study conducted by Sessa et al. [7], the researchers evaluated the results of a new method for treating knee JOCD in 20 patients. The methodology involved employing a collagen scaffold that did not contain any cells. Upon the final follow-up, it was seen that all scores exhibited notable enhancement, and MRI scans indicated complete filling or slight enlargement of the cartilage defect in 84.6% of the patients. Vannini et al. [8] monitored six patients with knee JOCD who had a treatment involving the transplantation of bone marrow-derived cells, platelet-rich plasma, bone marrow concentrate, and a hyaluronic acid scaffold in a single step. They showed that, in general, the patients saw a considerable decrease in symptoms at their final follow-up, enabling them to resume the majority of their everyday activities and sports. Furthermore, the newly formed tissue, assessed by MRI, completely occupied the whole volume of the osteochondral defects in every instance. According to a recent prospective study conducted by Russo et al. [9], a 13-year-old patient with JOCD of the patella experienced pain relief and improved function after one year of follow-up. Additionally, there was an improvement in cartilage volume and the structure of the injured area after two years of follow-up, thanks to the use of AD-MSCs.

Conclusions

Knee JOCD typically affects young and growing people. It is believed to predominantly harm the subchondral bone, leading to further damage to the articular cartilage. The precise cause of this condition is not known; however, several possibilities have been suggested, including repeated trauma, ischemia, hereditary predisposition, and anatomical variables. While radiographs are typically the initial imaging examination conducted for these individuals, they are unable to ascertain the stability or instability of the surface cartilage in the JOCD lesion. Consequently, MRI has gained importance as the preferred diagnostic technique for assessing the stability information of JOCD, which is crucial for establishing a treatment strategy. Conservative treatment is often advised for stable JOCD lesions with intact articular cartilage. Various surgical procedures are available for the treatment of stable lesions that do not heal with nonoperative therapy and unstable knee JOCD, with high percentages of successful healing.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ibrahim Akkawi

Acquisition, analysis, or interpretation of data: Ibrahim Akkawi, Hassan Zmerly, Maurizio Draghetti, Lamberto Felli

Drafting of the manuscript: Ibrahim Akkawi, Hassan Zmerly, Maurizio Draghetti, Lamberto Felli

Critical review of the manuscript for important intellectual content: Ibrahim Akkawi, Hassan Zmerly, Maurizio Draghetti, Lamberto Felli

Supervision: Lamberto Felli

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Nakayama H, Iseki T, Kambara S, Yoshiya S: Analysis of risk factors for poor prognosis in conservatively managed juvenile osteochondritis dissecans of the lateral femoral condyle. *Knee*. 2016, 23:950-4. [10.1016/j.knee.2016.09.021](#)
2. Erickson BJ, Chalmers PN, Yanke AB, Cole BJ: Surgical management of osteochondritis dissecans of the knee. *Curr Rev Musculoskelet Med*. 2013, 6:102-14. [10.1007/s12178-013-9156-0](#)
3. D'Angelo K, Kim P, Murnaghan ML: Juvenile osteochondritis dissecans in a 13-year-old male athlete: a case report. *J Can Chiropr Assoc*. 2014, 58:384-94.
4. Beck JJ, Sugimoto D, Micheli L: Sustained results in long-term follow-up of autologous chondrocyte implantation (ACI) for distal femur juvenile osteochondritis dissecans (JOCD). *Adv Orthop*. 2018, 2018:7912975. [10.1155/2018/7912975](#)
5. Eismann EA, Pettit RJ, Wall EJ, Myer GD: Management strategies for osteochondritis dissecans of the knee in the skeletally immature athlete. *J Orthop Sports Phys Ther*. 2014, 44:665-79. [10.2519/jospt.2014.5140](#)
6. Trinh TQ, Harris JD, Flannigan DC: Surgical management of juvenile osteochondritis dissecans of the knee. *Knee Surg Sports Traumatol Arthrosc*. 2012, 20:2419-29. [10.1007/s00167-012-1917-6](#)
7. Sessa A, Romandini I, Andriolo L, Di Martino A, Busacca M, Zaffagnini S, Filardo G: Treatment of juvenile knee osteochondritis dissecans with a cell-free biomimetic osteochondral scaffold: clinical and MRI results at mid-term follow-up. *Cartilage*. 2021, 13:1137S-47S. [10.1177/1947603520954500](#)
8. Vannini F, Battaglia M, Buda R, Cavallo M, Giannini S: "One step" treatment of juvenile osteochondritis dissecans in the knee: clinical results and T2 mapping characterization. *Orthop Clin North Am*. 2012, 43:237-44, vi. [10.1016/j.jocl.2012.02.003](#)
9. Russo A, Coco V, Zaffagnini S: The effect of autologous adipose derived mesenchymal stem cell therapy on juvenile osteochondritis dissecans of the patella: a case study. *J Surg Case Rep*. 2020, 2020:rjaa274. [10.1093/jscr/rjaa274](#)
10. Kumar V, Bhatnagar N, Lodhi JS: Grade I osteochondritis dissecans in a young professional athlete. *Indian J Orthop*. 2018, 52:344-52. [10.4103/ortho.IJOrtho_322_17](#)
11. Bausch L, Probst M, Fritsch L, Mehl J, Siebenlist S, Willinger L: Bilateral juvenile osteochondrosis dissecans in monozygotic twins: a case report. *J Orthop Surg Res*. 2024, 19:208. [10.1186/s13018-024-04683-2](#)
12. Bangert Y, Zarembowicz P, Engelleiter K, Gkarilas E, Schmitt H, Renkawitz T, Jaber A: Long-term outcome and athletic level following operative treatment for osteochondritis dissecans of the knee in pediatric and adolescent patients. *J Clin Med*. 2023, 12:4140. [10.3390/jcm12124140](#)
13. Yang JS, Bogunovic L, Wright RW: Nonoperative treatment of osteochondritis dissecans of the knee. *Clin Sports Med*. 2014, 33:295-304. [10.1016/j.csm.2013.11.003](#)
14. Kessler JL, Jacobs JC Jr, Cannamela PC, Shea KG, Weiss JM: Childhood obesity is associated with osteochondritis dissecans of the knee, ankle, and elbow in children and adolescents. *J Pediatr Orthop*. 2018, 38:e296-9. [10.1097/BPO.0000000000001158](#)
15. Deie M, Ochi M, Sumen Y, Kawasaki K, Adachi N, Yasunaga Y, Ishida O: Relationship between osteochondritis dissecans of the lateral femoral condyle and lateral menisci types. *J Pediatr Orthop*. 2006, 26:79-82. [10.1097/01.bpo.0000191554.34197.fd](#)
16. Hashimoto Y, Nishino K, Reid JB 3rd, et al.: Factors related to postoperative osteochondritis dissecans of the lateral femoral condyle after meniscal surgery in juvenile patients with a discoid lateral meniscus. *J Pediatr Orthop*. 2020, 40:e853-9. [10.1097/BPO.0000000000001636](#)
17. Chow RM, Guzman MS, Dao Q: Intercondylar notch width as a risk factor for medial femoral condyle osteochondritis dissecans in skeletally immature patients. *J Pediatr Orthop*. 2016, 36:640-4. [10.1097/BPO.0000000000000511](#)
18. Ishikawa M, Adachi N, Yoshikawa M, et al.: Unique anatomic feature of the posterior cruciate ligament in knees associated with osteochondritis dissecans. *Orthop J Sports Med*. 2016, 4:2325967116648138. [10.1177/2325967116648138](#)
19. Rupp MC, Hochberger F, Berthold DP, Muench LN, Imhoff AB, Siebenlist S, Willinger L: Tibiofemoral subluxation on radiograph as a predictor of location and size of osteochondritis dissecans lesions of the knee. *Orthop J Sports Med*. 2024, 12:23259671241232397. [10.1177/23259671241232397](#)
20. Jacobi M, Wahl P, Bouaicha S, Jakob RP, Gautier E: Association between mechanical axis of the leg and osteochondritis dissecans of the knee: radiographic study on 103 knees. *Am J Sports Med*. 2010, 38:1425-8. [10.1177/0363546509359070](#)
21. Wechter JF, Sikka RS, Alwan M, Nelson BJ, Tompkins M: Proximal tibial morphology and its correlation with osteochondritis dissecans of the knee. *Knee Surg Sports Traumatol Arthrosc*. 2015, 23:3717-22. [10.1007/s00167-014-3289-6](#)
22. Cavaignac E, Perroncel G, Thépaut M, Vial J, Accadbled F, De Gauzy JS: Relationship between tibial spine size and the occurrence of osteochondritis dissecans: an argument in favour of the impingement theory. *Knee Surg Sports Traumatol Arthrosc*. 2017, 25:2442-6. [10.1007/s00167-015-3907-y](#)
23. Camathias C, Hirschmann MT, Vavken P, Rutz E, Brunner R, Gaston MS: Meniscal suturing versus screw fixation for treatment of osteochondritis dissecans: clinical and magnetic resonance imaging results. *Arthroscopy*. 2014, 30:1269-79. [10.1016/j.arthro.2014.05.010](#)
24. Maier GS, Lazovic D, Maus U, Roth KE, Horas K, Seeger JB: Vitamin D deficiency: the missing etiological factor in the development of juvenile osteochondrosis dissecans?. *J Pediatr Orthop*. 2019, 39:51-4. [10.1097/BPO.0000000000000921](#)
25. Hussain WM, Hussain HM, Hussain MS, Ho SS: Human growth hormone and the development of osteochondritis dissecans lesions. *Knee Surg Sports Traumatol Arthrosc*. 2011, 19:2108-10. [10.1007/s00167-010-1370-3](#)
26. McElroy MJ, Riley PM, Tepolt FA, Nasreddine AY, Kocher MS: Catcher's knee: posterior femoral condyle juvenile osteochondritis dissecans in children and adolescents. *J Pediatr Orthop*. 2018, 38:410-7. [10.1097/BPO.0000000000000839](#)
27. Price MJ, Tuca M, Nguyen J, Silberman J, Luderowski E, Uppstrom TJ, Green DW: Juvenile osteochondritis dissecans of the trochlea: a cohort study of 34 trochlear lesions associated with sporting activities that load the patellofemoral joint. *J Pediatr Orthop*. 2020, 40:103-9. [10.1097/BPO.0000000000001174](#)

28. Kessler JJ, Nikizad H, Shea KG, Jacobs JC Jr, Bechuk JD, Weiss JM: The demographics and epidemiology of osteochondritis dissecans of the knee in children and adolescents. *Am J Sports Med.* 2014, 42:320-6. [10.1177/0363546513510390](https://doi.org/10.1177/0363546513510390)
29. Kocher MS, Tucker R, Ganley TJ, Flynn JM: Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med.* 2006, 34:1181-91. [10.1177/0363546506290127](https://doi.org/10.1177/0363546506290127)
30. Lee CS, Larsen CG, Marchwiany DA, Chudik SC: Extra-articular, intraepiphyseal drilling for osteochondritis dissecans of the knee: characterization of a safe and reproducible surgical approach. *Orthop J Sports Med.* 2019, 7:2325967119830397. [10.1177/2325967119830397](https://doi.org/10.1177/2325967119830397)
31. Conrad JM, Stanitski CL: Osteochondritis dissecans: Wilson's sign revisited. *Am J Sports Med.* 2003, 31:777-8. [10.1177/03635465030310052301](https://doi.org/10.1177/03635465030310052301)
32. Haeri Hendy S, de Sa D, Ainsworth K, Ayeni OR, Simunovic N, Peterson D: Juvenile osteochondritis dissecans of the knee: does magnetic resonance imaging instability correlate with the need for surgical intervention?. *Orthop J Sports Med.* 2017, 5:2325967117738516. [10.1177/2325967117738516](https://doi.org/10.1177/2325967117738516)
33. Roßbach BP, Paulus AC, Niethammer TR, et al.: Discrepancy between morphological findings in juvenile osteochondritis dissecans (OCD): a comparison of magnetic resonance imaging (MRI) and arthroscopy. *Knee Surg Sports Traumatol Arthrosc.* 2016, 24:1259-64. [10.1007/s00167-015-3724-3](https://doi.org/10.1007/s00167-015-3724-3)
34. De Smet AA, Ilahi OA, Graf BK: Untreated osteochondritis dissecans of the femoral condyles: prediction of patient outcome using radiographic and MR findings. *Skeletal Radiol.* 1997, 26:463-7. [10.1007/s002560050267](https://doi.org/10.1007/s002560050267)
35. Heywood CS, Benke MT, Brindle K, Fine KM: Correlation of magnetic resonance imaging to arthroscopic findings of stability in juvenile osteochondritis dissecans. *Arthroscopy.* 2011, 27:194-9. [10.1016/j.arthro.2010.07.009](https://doi.org/10.1016/j.arthro.2010.07.009)
36. Dipaola JD, Nelson DW, Colville MR: Characterizing osteochondral lesions by magnetic resonance imaging. *Arthroscopy.* 1991, 7:101-4. [10.1016/0749-8063\(91\)90087-e](https://doi.org/10.1016/0749-8063(91)90087-e)
37. Samora WP, Chevillet J, Adler B, Young GS, Klingele KE: Juvenile osteochondritis dissecans of the knee: predictors of lesion stability. *J Pediatr Orthop.* 2012, 32:1-4. [10.1097/BPO.0b013e31823d8312](https://doi.org/10.1097/BPO.0b013e31823d8312)
38. Guhl JF: Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res.* 1982, 65:74.
39. O'Connor MA, Palaniappan M, Khan N, Bruce CE: Osteochondritis dissecans of the knee in children. A comparison of MRI and arthroscopic findings. *J Bone Joint Surg Br.* 2002, 84:258-62. [10.1302/0301-620x.84b2.11823](https://doi.org/10.1302/0301-620x.84b2.11823)
40. Kijowski R, Blankenbaker DG, Shinki K, Fine JP, Graf BK, De Smet AA: Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. *Radiology.* 2008, 248:571-8. [10.1148/radiol.2482071234](https://doi.org/10.1148/radiol.2482071234)
41. Hefti F, Beguirstain J, Krauspe R, et al.: Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B.* 1999, 8:231-45.
42. Brittberg M, Winalski CS: Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am.* 2003, 85-A Suppl 2:58-69. [10.2106/00004623-200300002-00008](https://doi.org/10.2106/00004623-200300002-00008)
43. Ewing JW, Voto SJ: Arthroscopic surgical management of osteochondritis dissecans of the knee. *Arthroscopy.* 1988, 4:37-40. [10.1016/s0749-8063\(88\)80010-0](https://doi.org/10.1016/s0749-8063(88)80010-0)
44. Pascual-Garrido C, Moran CJ, Green DW, Cole BJ: Osteochondritis dissecans of the knee in children and adolescents. *Curr Opin Pediatr.* 2013, 25:46-51. [10.1097/MOP.0b013e3182835adb5](https://doi.org/10.1097/MOP.0b013e3182835adb5)
45. Krause M, Hapfelmeier A, Möller M, Amling M, Bohnndorf K, Meenen NM: Healing predictors of stable juvenile osteochondritis dissecans knee lesions after 6 and 12 months of nonoperative treatment. *Am J Sports Med.* 2013, 41:2384-91. [10.1177/0363546513496049](https://doi.org/10.1177/0363546513496049)
46. Hughes JA, Cook JV, Churchill MA, Warren ME: Juvenile osteochondritis dissecans: a 5-year review of the natural history using clinical and MRI evaluation. *Pediatr Radiol.* 2003, 33:410-7. [10.1007/s00247-003-0876-y](https://doi.org/10.1007/s00247-003-0876-y)
47. Gunton MJ, Carey JL, Shaw CR, Murnaghan ML: Drilling juvenile osteochondritis dissecans: retro- or transarticular?. *Clin Orthop Relat Res.* 2013, 471:1144-51. [10.1007/s11999-011-2237-8](https://doi.org/10.1007/s11999-011-2237-8)
48. Webb JE, Lewallen LW, Christophersen C, Krych AJ, McIntosh AL: Clinical outcome of internal fixation of unstable juvenile osteochondritis dissecans lesions of the knee. *Orthopedics.* 2013, 36:e1444-9. [10.3928/01477447-20131021-30](https://doi.org/10.3928/01477447-20131021-30)
49. Tabaddor RR, Banffy MB, Andersen JS, McFeely E, Ogunwole O, Micheli LJ, Kocher MS: Fixation of juvenile osteochondritis dissecans lesions of the knee using poly 96L/4D-lactide copolymer bioabsorbable implants. *J Pediatr Orthop.* 2010, 30:14-20. [10.1097/BPO.0b013e3181c6318c](https://doi.org/10.1097/BPO.0b013e3181c6318c)
50. Komnos G, Iosifidis M, Papageorgiou F, Melas I, Metaxiotis D, Hantes M: Juvenile osteochondritis dissecans of the knee joint: midterm clinical and MRI outcomes of arthroscopic retrograde drilling and internal fixation with bioabsorbable pins. *Cartilage.* 2021, 13:1228S-36S. [10.1177/19476035211003325](https://doi.org/10.1177/19476035211003325)
51. Gudas R, Simonaityte R, Cekanauskas E, Tamosiūnas R: A prospective, randomized clinical study of osteochondral autologous transplantation versus microfracture for the treatment of osteochondritis dissecans in the knee joint in children. *J Pediatr Orthop.* 2009, 29:741-8. [10.1097/BPO.0b013e3181b8f6c7](https://doi.org/10.1097/BPO.0b013e3181b8f6c7)
52. Miniaci A, Tytherleigh-Strong G: Fixation of unstable osteochondritis dissecans lesions of the knee using arthroscopic autogenous osteochondral grafting (mosaicplasty). *Arthroscopy.* 2007, 23:845-51. [10.1016/j.arthro.2007.02.017](https://doi.org/10.1016/j.arthro.2007.02.017)
53. Miura K, Ishibashi Y, Tsuda E, Sato H, Toh S: Results of arthroscopic fixation of osteochondritis dissecans lesion of the knee with cylindrical autogenous osteochondral plugs. *Am J Sports Med.* 2007, 35:216-22. [10.1177/0363546506294360](https://doi.org/10.1177/0363546506294360)
54. Moriya T, Wada Y, Watanabe A, Sasho T, Nakagawa K, Mainil-Varlet P, Moriya H: Evaluation of reparative cartilage after autologous chondrocyte implantation for osteochondritis dissecans: histology, biochemistry, and MR imaging. *J Orthop Sci.* 2007, 12:265-73. [10.1007/s00776-007-1111-8](https://doi.org/10.1007/s00776-007-1111-8)
55. Krishnan SP, Skinner JA, Carrington RW, Flanagan AM, Briggs TW, Bentley G: Collagen-covered autologous chondrocyte implantation for osteochondritis dissecans of the knee: two- to seven-year results. *J Bone Joint Surg Br.* 2006, 88:203-5. [10.1302/0301-620X.88B2.17009](https://doi.org/10.1302/0301-620X.88B2.17009)
56. Friederichs MG, Greis PE, Burks RT: Pitfalls associated with fixation of osteochondritis dissecans fragments

using bioabsorbable screws. Arthroscopy. 2001, 17:542-5. [10.1053/jars.2001.22397](https://doi.org/10.1053/jars.2001.22397)