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Juvenile osteochondritis dissecans of the knee: current trends in diagnostic and treatment

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SUMMARY

Juvenile osteochondritis dissecans (JOCD) is an acquired condition of the joint that affects the articular surface and the subchondral bone in skeletally immature patients. Natural history is poorly characterized based on limited information from retrospective case series. Although JOCD generally presents as a stable lesion amenable to nonoperative treatment or minimally-invasive drilling, unstable forms can require a more aggressive approach. Early diagnosis and treatment is essential to prevent further cartilage destruction, and preserve joint function. This article review the most recent literature available, and focuses on the pathophysiology, diagnosis, and treatment of JOCD of the knee.
Evidence level: V

Key words: Osteochondritis, juvenile, treatment.
(Rev Mex Ortop Ped 2018; 1:34-43)

RESUMEN

La osteocondritis disecante juvenil (OCDJ) es una condición adquirida de la articulación que afecta a la superficie articular y el hueso subcondral en pacientes esqueléticamente inmaduros. La historia natural ha sido pobremente caracterizada basada en información limitada de series de casos retrospectivas. La OCDJ se puede presentar como una lesión estable que puede ser tratada en forma no quirúrgica, o en ocasiones con tratamientos mínimamente invasivos como la perforación artroscópica. Las formas más inestables pueden requerir un tratamiento más agresivo. El diagnóstico y tratamiento tempranos son esenciales para prevenir la destrucción del cartílago y preservar la función articular. Este artículo es una revisión de la literatura más reciente enfocándose en la fisiopatología, el diagnóstico y el tratamiento de la OCDJ.
Nivel de evidencia: V

Palabras clave: Osteocondritis, juvenil, tratamiento.
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INTRODUCTION

Osteochondritis dissecans (OCD) is an acquired condition of the joint that affects the articular surface and the subchondral bone of the knee, elbow, and ankle. The juvenile form of the disease (JOCD) presents in those aged 5 to 16 years with open growth plates.[1] The causes of OCD are unknown, however repetitive trauma, inflammation, accessory centers of ossification, ischemia, and genetic factors has been proposed. Patients with JOCD present with vague pain and occasionally, mechanical symptoms. When OCD affects the knee, the most common location is within the lateral aspect of the medial femoral condyle. The goal of treatment is to promote healing in the subchondral bone and prevent chondral collapse, subsequent fracture, and crater formation. The treatment depends on skeletal maturity of the patient, as well as the size, stability, and location of the lesion.

The aim of this article is to provide a summary on the current literature relating to pathophysiology, diagnosis, and treatment of JOCD of the knee.

Objectives:

- Review the pathophysiology and presentation of JOCD of the knee.
- Discuss clinical presentation, imaging evaluation, and surgical indications.
- Discuss expected outcomes and potential complications.

EPIDEMIOLOGY

The exact incidence of JOCD of the knee is unknown. A Swedish study by Lindén et al.[2] estimated the overall prevalence of knee OCD to be 15 to 29 per 100,000 patients each year. Osteochondritis dissecans most

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frequently presents between 13 and 21 years of age. Incidence rises from 6.8 per 100,000 in those between six and 11 years to 11.2 per 100,000 in the 12- to 16-year age group.[3] Osteochondritis dissecans seems to affect males more commonly than females (between 2:1 and 3:1). However, as females participate in sports there has been an increased prevalence among girls.

JOCD of the knee occurs most frequently in the classic location of the posterolateral aspect of the medial femoral condyle. In a large multicenter study,[4] 77% of lesions affected the medial femoral condyle (51% are on the lateral aspect, 19% central and 7% medial), 17% in the lateral femoral condyle, 7% in the patella, 1% trochlear lesions, and only 0.2% arose from the tibial plateau.[5]

ETIOLOGY

The causes of OCD are unknown. Theories range from abnormal vascular anatomy (leading to ischemic injury of the bone), abnormal ossification of the epiphysis, trauma, endocrine imbalances, genetic predisposition or some combination of the above. Previous authors have suggested a genetic link in monozygotic twins and syndromic patients.[6] Other researchers have described familial cases of OCD lesions associated with short stature and multiple lesion sites.[7] In a recent publication,[8] the proportion of patients with a positive family history of OCD was 14%. Although a small subset of patients have an inherited form of JOCD, non-familial OCD is most prevalent.

Recent unpublished data from the ROCK study group has provided new MRI findings to support previous theories regarding JOCD formation through aberrant development of only a portion of the epiphyseal growth plate.[9] Abnormal epiphyseal endochondral ossification may occur after a particular acute or repetitive insult, leading to a slowly evolving lesion as the patient ages. Evidence from T2 fat saturation sequences has helped describe two potential scenarios. The first is a permanent cessation of ossification after insult that leads to a completely cartilaginous OCD lesion without endochondral ossification. The second scenario involves temporary cessation of ossification that allows for future partial or complete normal ossification with time.[9,10]

CLINICAL MANIFESTATIONS

Symptoms are variable and range from asymptomatic to significant pain and locking. JOCD typically presents with poorly localized knee pain in highly active patients. The pain is worsened by exercise. Crepitus, catching, or locking of the joint may occur during the later stages. The diagnosis can frequently be made by clinical findings and judicious use of imaging.

The physical examination should include careful inspection of the knee, palpation for point tenderness, assessment of joint effusion, range-of-motion, evaluation of limb alignment, and associated injuries (ligaments/meniscus). The Wilson test is of limited diagnostic value and should not be used to rule in or out the diagnosis of OCD.[4] A routine thorough physical examination of the hip should be performed to rule out hip pathology, which can commonly present with knee symptoms secondary to referred pain.

IMAGING

Anterior–posterior (AP), lateral, tunnel (or notch), and sunrise radiographs are recommended in patients suspected to have JOCD *(Figure 1)*. Tunnel view of the knee often provide the best visualization of the lesions

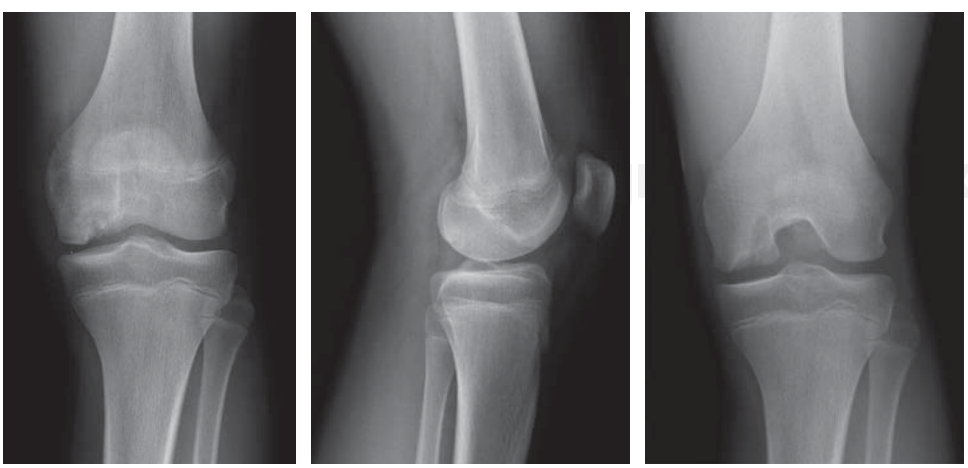


Figure 1.

Anteroposterior, lateral, and tunnel view radiograph of a 13-year-old rugby player with a lesion in the medial femoral condyle that affects the weight-bearing area of the right knee.

located in the femoral condyle. Characteristic findings include a well-circumscribed area of subchondral bone separated by a crescent-shaped sclerotic radiolucent outline of the fragment. Bilateral radiographic knee evaluation is recommended for all patients found to have JOCD. In a large case series,[11] a 29% incidence of bilateral disease was found, and almost 40% were asymptomatic upon presentation.

MRI is a valuable tool for diagnosis as well as for monitoring the progression and/or the healing of these lesions. MRI has the capability of assessing the surrounding cartilage and subchondral bone that is not seen on conventional radiographs, and has superior detail and definition of structures within the knee joint *(Figure 2)*. Although MRI is the best imaging modality for JOCD with a high diagnostic sensitivity (approximately 100 percent), it is not possible to predict accurately the stability of the fragment. Heywood et al[12] reported a specificity of 15% for diagnosing fragment instability. In a recent study from Germany,[13] the authors found a poor correlation between preoperative MRI and arthroscopy morphological findings (59.6% of all patients with an OCD of the femoral condyle). Both studies agree that MRI should not be used in isolation to determine lesion stability in young patients with juvenile OCD. Technological improvements to MRI practices, such as the use of higher magnet strengths of 3.0 T, may provide superior diagnostic performance strategies in the future.[14]

DIFFERENTIAL DIAGNOSIS

Several conditions have clinical features that overlap with knee JOCD. Most can be identified by a careful history, physical examination and radiographs/MRI. The differential diagnosis includes: torn meniscus, symptomatic discoid meniscus, osteochondral fracture, patelofemoral syndrome, and symptomatic medial plicae.

CLASSIFICATIONS

There are several classification systems for OCD, including those based on plain radiographs,[5] MRIs[15] and arthroscopic findings[16] *(Table I)*. No system has been universally accepted.[17] The Research in Osteochondritis of the Knee (ROCK) study group

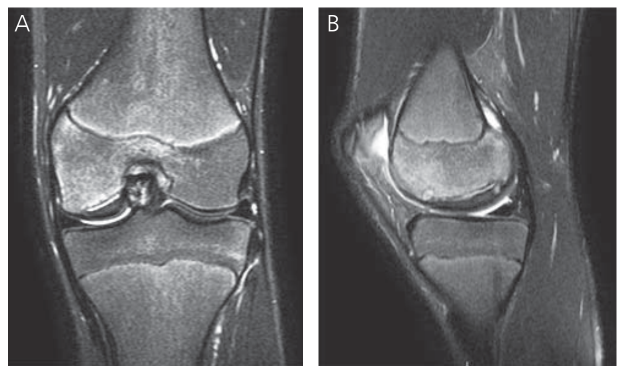


Figure 2. *Magnetic resonance image (of the same patient as in* ***figure 1). A)*** *T2 Coronal image of the knee of an osteochondritis dissecans lesion of the medial femoral condyle; note the high signal between the osteochondral fragment and subchondral bone, and subchondral bone marrow edema.* ***B)*** *T2 Sagittal image through the medial femoral condyle shows the presence of a cyst-like lesion associated with a large juvenile osteochondritis dissecans lesion.*

Table I. Proposed classification systems for osteochondritis dissecans.

XR		MRI		Arthroscopy	
Berndt and Harty[5]		Dipaola[15]		Guhl[16]	
Stage 1	Small area, compression subchondral bone	Type I	Thickening of articular cartilage, but no break	Type I	Softening and irregularity of cartilage but no fragment
Stage 2	Partially detached OCD fragment	Type II	Breached articular cartilage, low signal rim behind fragment indicating attachment	Type II	Breached articular cartilage, with the fragment not displaceable
Stage 3	Fully detached OCD fragment, still in underlying crater	Type III	Breached articular cartilage, with high signal T2 changes behind fragment suggesting fluid around lesion	Type III	Definable fragment, partially attached but displaceable (flap lesion)
Stage 4	Complete detachment/loose body	Type IV	Loose body and defect of articular surface	Type IV	Loose body and defect of articular surface

Abbreviations: XR = plain radiographs, MRI = magnetic resonance image.

developed recently a novel classification system for arthroscopic evaluation of OCD of the knee that includes 6 arthroscopic categories-3 immobile types and 3 mobile types *(Figure 3)*. This comprehensive arthroscopic classification system demonstrated excellent intra- and interobserver reliability.[18]

TREATMENT

Management of knee JOCD still remains a controversial topic. A recent Clinical Practice Guidelines published by the American Academy of Orthopaedic Surgeons (AAOS) was unable to make any recommendations graded as «strong» regarding treatment.[19] The best evidence available to date is limited to retrospective case series, and expert opinions, which limits firm conclusions to be drawn.

Conservative treatment

Conservative treatment should be the primary approach for stable JOCD of the knee. Although there is a lack of evidence for specific non-operative treatments, most authors recommend at least 3-6 months prior to the decision for surgical treatment. Nonoperative treatment options include: immobilization (casting, bracing, splinting, unloader brace), limited weight-bearing, and activity restriction. There is controversy regarding the duration and timing of these interventions. The AAOS guidelines were unable to recommend any particular nonoperative treatment regimen.[19]

The author applies the 3-phase nonoperative management protocol described by Kocher et al.[20] The first phase involves knee immobilization for 4 to

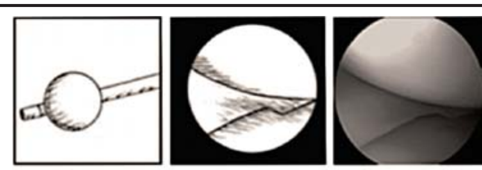
	Type and description	Diagrams
Immobile lesions	Cue ball: No abnormality detectable arthroscopically	
	Shadow: Cartilage is intact and subtly demarcated (possibly under low light)	
	Wrinkle in the rug: Cartilage is demarcated with a fissure, buckle, and/or wrinkle	
Mobile lesions	Locked door: Cartilage fissuring at periphery, *unable* to hinge open	
	Trap door: Cartilage fissuring at periphery, *able* to hinge open	
	Crater: Exposed subchondral bone defect	

Figure 3. *ROCK arthroscopic classification of JOCD.*

6 weeks with crutch-protected, partial weightbearing gait. At the end of this period, the child should be pain free, and repeat radiographs should be obtained. In phase 2 (weeks 6-12), weightbearing as tolerated is permitted without immobilization. A rehabilitation program is initiated emphasizing knee range of motion and low-impact quadriceps and hamstring strengthening exercises. Sports and repetitive-impact activities are restricted. If there are radiographic and clinical signs of healing at 3 to 4 months after the initial diagnosis, phase 3 can begin. This phase includes supervised initiation of running, jumping, and cutting sports readiness activities. A gradual return to sports with increasing intensity is allowed in the absence of knee symptoms. An MRI is repeated in phase 3 to assess healing.

Wall et al.[21] reported a series of patients with stable JOCD of the knee treated nonoperatively. Treatment consisted of an initial 6-week period of weight-bearing immobilization in a cylinder cast. If the lesion showed reossification on radiographs, casting was discontinued. If reossification was not present, the patients were allowed 3 to 7 days out of the cast to regain range of motion, followed by casting for an additional 4 to 6 weeks. After casting, patients were placed in an unloader brace and restricted from running, jumping, and sports. During this phase, patients were radiographed every 6 to 8 weeks and activity was slowly advanced, as long as radiographs showed progression of healing. Return to full activity was allowed after complete reossification was demonstrated on radiographs. After 6 months of nonoperative treatment only seven of 47 (15%) knees had completely reossified, whereas 16 of 47 (34%) had no progression towards healing. Krause et al.[22] showed that after 6 months only 26% of the 76 lesions had progressed toward healing after nonoperative treatment. Potential causes of a higher failure rate in Krause´s series might have been the images used for outcome measures (MRI rather than radiography), and the inclusion for treatment of more severe lesions. Samora et al.[23] have reported that lesions in atypical locations, such as the non–weight-bearing portion of the lateral femoral condyle, are more likely to be unstable and associated with lower healing rates with nonoperative treatment. Other authors have reported that presenting symptoms of effusion or mechanical features, larger lesion size and the presence and extent of sublesional sclerosis on radiographs are predictive of nonhealing at six months.[21,24]

Beyond a consensus that nonoperative treatment should be used for at least 3-6 months in stable lesions, there is little agreement on wich regimen is more effective. Future research in this area is required to compare different treatment protocols.

Surgical options

Surgical treatment to promote healing is suggested in stable (immobile) lesions not responding to an initial course of nonoperative therapy, and unstable (mobile) lesions. Surgery for osteochondritis dissecans may include drilling, internal fixation, and salvage procedures. The authors' preferred treatment algorithm for osteochondritis dissecans (OCD) of the knee is based on lesion stability, skeletal maturity, and clinical symptoms *(Figure 4)*.

Drilling

Surgical treatment for stable lesions with intact articular cartilage involves drilling the subchondral bone with the intention of stimulating vascular ingrowth and subchondral bone healing. Arthroscopically confirmed stable JOCD lesions can be drilled either in a transarticular or retroarticular fashion. The authors' preferred technique is retroarticular drilling with fluoroscopic guidance.[25] This technique spares the articular surface and physes by drilling through the affected condyle. Retro-articular drilling and bone grafting using a bone marrow biopsy needle has also been described.[26,27] On the other hand, transarticular drilling penetrates the lesion through the articular cartilage. Concerns with this technique involve the uncertain long-term implications for joint surface damage created by articular cartilage drill sites.

Surgical technique

Surgery is performed under general or spinal anesthesia using a thigh tourniquet. Diagnostic arthroscopy is performed to confirm intact articular cartilage. The arthroscopy equipment is then removed from the joint and the C-arm fluoroscopy machine is positioned. AP, tunnel, and true lateral views are obtained, identifying the area of the JOCD lesion. Under fluoroscopic guidance, a 1.6 mm K-wire is inserted percutaneously using free-hand technique at a level below the physis toward the center of the lesion just beneath the articular cartilage in a retrograde fashion. Multiple parallel K-wires are placed into different positions within the lesion using the first k-wire as a guide *(Figure 5)*. It is important to

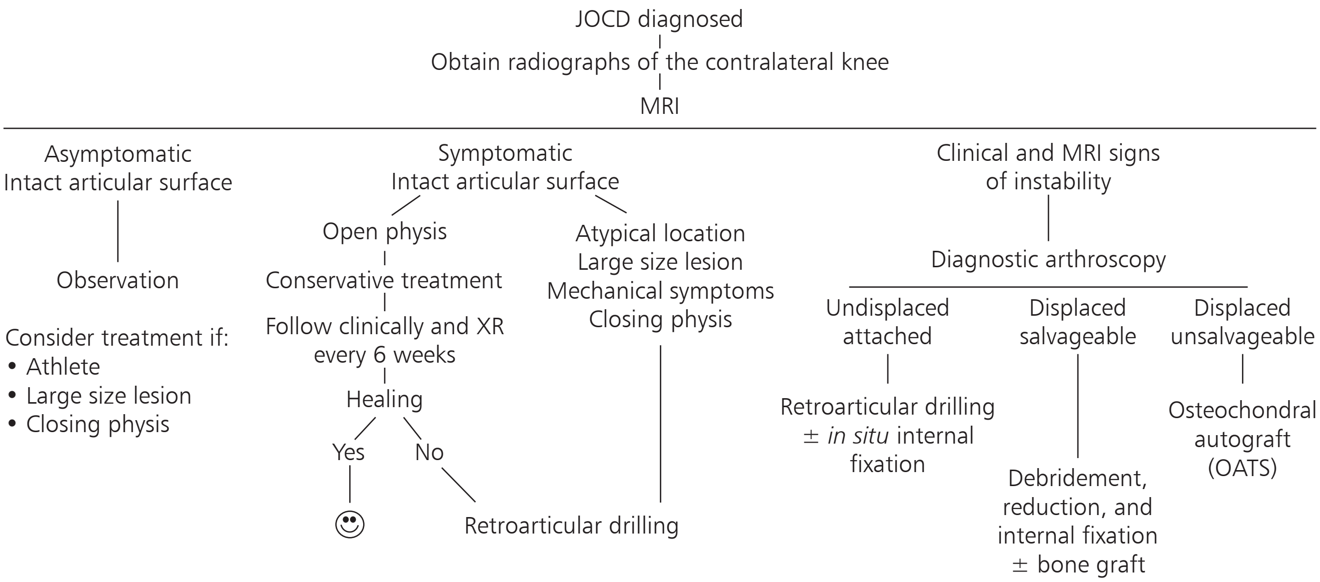


Figure 4. *Authors' preferred treatment algorithm for juvenile osteochondritis dissecans (JOCD) of the knee.*

check the direction of the guide wire under AP, tunnel and lateral fluoroscopic views for reaching the lesion accurately. After drilling, the tourniquet is released, the arthroscopic portals are closed with subcuticular sutures, and a dressing is then applied.

Patients immediately begin a series of active motion exercises of the knee after surgery. Physical therapy is performed from the second week after surgery. Full weight-bearing is permitted 6 weeks postoperatively, and sporting activities, after 4-6 months. Return to play is allowed once the lesion has healed and quadriceps strength has returned to within normal limits.

Outcomes of OCD drilling are favorable in most cases. Baroni and Masquijo[25] reported the clinical outcomes of 21 skeletally immature patients (21 knees) treated with retroarticular drilling. There were 14 boys and 7 girls. The lesions were located in the medial femoral condyle in 16 knees and in the lateral femoral condyle in 5 knees. The average age at the time of surgery was 12 years, 4 months (range, 9-15 years). There were no intraoperative or postoperative complications. The average follow-up was 5.7 years. Nineteen patients (90.5%) showed complete radiographic healing, and full return to activities. The remaining two patients had delayed consolidation and partial fragment detachment that required internal fixation. Several authors[28-30] have reported a high rate of healing and low complication rates using either transarticular or retroarticular drilling modalities. Gunton et al.[31] systematically reviewed the short-term clinical outcomes of retroarticular and transarticular drilling of stable OCD lesions. Twelve studies met inclusion criteria, 111 lesions were treated with retroarticular drilling, and 94 transarticular.

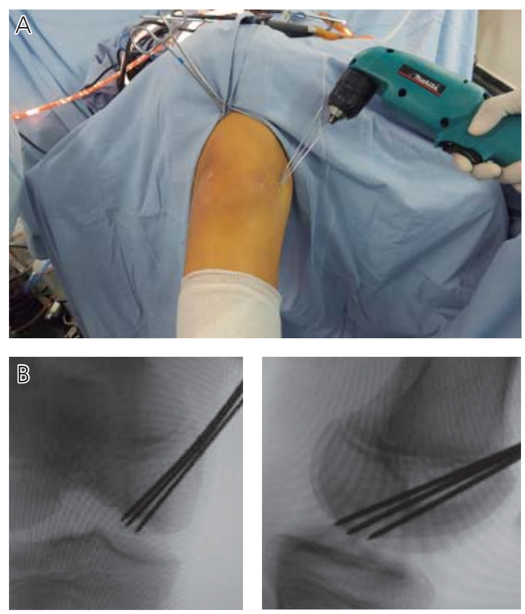


Figure 5. *Retroarticular drilling.* ***A)*** *Under fluoroscopic guidance, multiple 1.6 mm Kirschner-wires are placed percutaneously using free-hand technique at a level below the physis, and directed obliquely, down through the femoral condyle in a retrograde fashion.* ***B)*** *Accurate placement of the k-wires is checked under different fluoroscopic views.*

Both techniques result in comparable radiographic healing at 86 and 91%, respectively occurring at a mean of 4 to 6 months, with no reported complications.

Internal fixation

If there is any doubt about stability, internal fixation is necesary in addition to drilling. If accessible (trapdoor lesions), the fibrous tissue from base of the lesion and bony surface of the flap are debrided with a curette or arthroscopic shaver. If there is a resultant bone void, bone grafting from the proximal tibia or iliac crest is performed. Fixation can be accomplished with multiple metal or bioabsorbable devices.[32-35] Cannulated screws, Herbert screws, bone pegs, and metal staples have been used. Disadvantages with metallic implants include MRI interference, and the requirement of a second surgery to remove many of these devices.[36] Metallic implants have been associated with many complications such as migration, breakage, and loosening.[16] These concerns have led to the development of bioabsorbable implants. Bioabsorbable devices have been made from polyglycolic acid (PGA) or polylactic acid (PLA). PGA has a rapid degradation rate, reportedly absorbing in 3 months with high incidence of foreign body reactions.[35] PLA, which was later introduced in response to problems encountered with PGA devices, can take as long as six years to absorb, and may place opposing cartilage at risk for damage by the implant.[38,39] Most recent devices comprised both PGA and PLA copolymer with the aim of maximizing their beneficial effects while minimizing the inflammatory reaction elicited from degradation products[36] *(Figure 6)*. In most cases, fixation is accomplished with two to four of these implants, to impart compression and rotational stability to the fragment, and the lengths are chosen preoperatively to avoid violating the growth plate (usually 20 or 25 mm in length). Tabaddor et al.[36] reported the outcomes of 24 patients (24 knees) with unstable OCD lesions of the knee that were treated with SmartNails. The mean age at the time of surgery was 14.4 years. The mean followup was 39.6 months. Plain films at an average of 19.2 months postoperatively revealed interval healing in nine patients, no significant change in one patient, complete healing in 13 patients, and loose bodies with no interval healing in one patient. Twenty-two of 24 (91.7%) patients had good-to-excellent outcomes. More recently, a study from Japan reported improved clinical outcomes and 97% radiographic healing rate at a mean of 3.3 years of follow-up.[40]

Autogenous osteochondral plugs have recently been presented as a biologic alternative to the use of hardware. The plugs provide bone graft as well as fixation of the lesion. Three studies[41-43] reported that biological fixation provided healing of the osteochondral fragment with promising results.

Salvage techniques

When the progeny fragment has comminuted into multiple small fragments or is incongruous with the donor site, or the articular cartilage is excessively deteriorated, primary fixation may not be the most viable option.[44] There are several salvage techniques for full-thickness defects, such as marrow stimulation (microfractures), osteochondral autograft (OATS), fresh osteochondral allograft, and autologous chondrocyte implantation (ACI). This is a very unusual scenario for the pediatric-adolescent

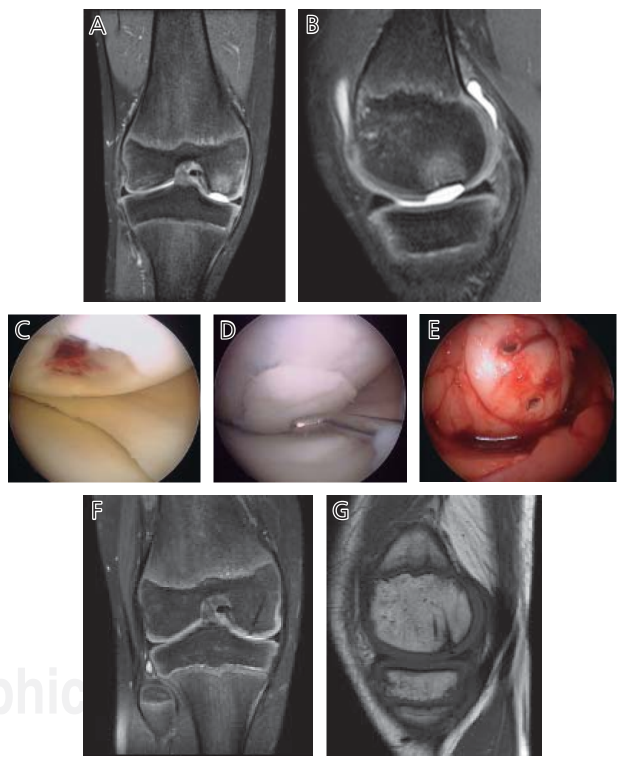


Figure 6. *Internal fixation.* ***A)*** *Coronal, and* ***B)*** *Sagittal T2 image MRI of the knee of an exposed subchondral bone defect of the medial femoral condyle in a 12 years-old basketball player,* ***C)*** *arthroscopic image of the defect,* ***D-E)*** *arthroscopic image repositioning the pure chondral fragment and fixation with two bioabsorbable SmartNails.* ***F-G)*** *MRI at 6 months shows complete healing.*

population, and literature is scarce on clinical outcome data.

Marrow stimulation techniques such as microfracture involve breaching the subchondral bone to allow the influx of pluripotent stem cells from the marrow into the osteochondral defect, resulting in fibrocartilage formation.[45] Microfracture is indicated in patients with a localized small cartilage defect. OATS technique transfers both articular cartilage and subchondral bone from a non-weight-bearing area of the knee to the site of the defect, and is most effective in lesions smaller than 2.5 cm^2.[46] Gudas et al.[47] randomized 50 children with a mean age of 14.3 years (12 to 18) to either microfracture or OATS for treatment of femoral condylar JOCD. At one year, both groups had good or excellent results in their functional and objective assessment, but at 4.2 years, the OATS group maintained an 83% good or excellent result, while the microfracture group dropped to 63%. Failure rates were 41% in the microfracture group and 0% in the OATS group, with an inverse relationship between defect size and outcome in the microfracture group, without a similar relationship in the OATS group. Only 14% of patients in the microfracture group returned to their preinjury level at 4.2 years versus 81% in the OATS group.

For larger lesions, osteochondral allograft transplantation procedures may be used. Advantages of this approach include the avoidance of donor-site morbidity and the ability to address large lesions with a single operation, concerns over the phenomenon of creep substitution and long-term maintenance of graft incorporation make additional follow-up studies critical to more widespread adoption of the procedure.[48]

ACI is a two-stage cellularly based autograft technique.[49] The first stage involves an arthroscopic biopsy from healthy cartilage in the non-weight-bearing region of the intercondylar notch. These cells are grown *in vitro* over 4-6 weeks, at which point the patient returns for implantation. During this procedure, the calcified cartilage is removed, and the lesion is debrided to stable vertical walls. The defect is covered with a periosteal patch from the proximal tibia or a synthetic collagen membrane that is sutured using interrupted 5-0 or 6-0 Vicryl sutures to the healthy edges of the debrided defect. Fibrin glue is then used to seal the edges, and the cultured cells are injected beneath the patch. One edge is left open until the cells are injected. The remaining defect is then securely closed with sutures and glue. Several clinical studies have reported promising results. Mithofer et al.[50] reported 96% good to excellent outcomes at the mid-term in adolescent patients undergoing ACI for OCD. Similarly, 96% of patients also returned to high impact sports and 60% returned to a level equal or higher than prior to their knee injury. Improved results were seen in patients with shorter duration of symptoms (< 12 months) and fewer surgeries. A recent systematic review of ACI on 115 adolescents (5 studies) who underwent ACI showed an average improvement of outcome scores near 40%. Mean patient age was 16.2 years (range, 11 to 21 years). Follow-up ranged from 12 to 74 months (mean, 52.3 months). Mean defect size was 5.3 cm^2 (range, 0.96 to 14 cm^2). Graft hypertrophy was the most common complication (7%).[51]

Future directions - The role of the ROCK study group

The ROCK study group was founded in 2010 to improve the outcomes of patients with knee OCD through multicenter research. Ongoing work of the ROCK study group includes development of a detailed and validated MRI classification system, multicenter randomized trial of transarticular versus retroarticular drilling of stable OCDs in skeletally immature patients, and multicenter prospective cohort of conservative and surgical treatment of knee OCD.[52]

CONCLUSIONS

Diagnosis of juvenile osteochondritis dissecans of the knee should be considered in young, active patients who have knee pain. Early diagnosis and treatment is essential to prevent cartilage destruction, and preserve joint function. Treatment should be individualized, based on skeletal maturity of the patient, as well as the size, stability, and location of the lesion. Although there is limited high quality literature surrounding the optimal management and prognosis of juvenile OCD of the knee, pediatric orthopaedic surgeons can draw upon current best practices outlined by the AAOS and ROCK organizations. Conservative treatment should be the first-line treatment for stable (immobile) lesions. Surgical treatment for stable lesions with intact articular cartilage involves drilling the subchondral bone aiming to stimulate vascular ingrowth and subchondral bone healing. If the lesion is unstable (mobile), fixation is indicated. Every attempt should be made to retain the osteochondral fragment when possible. If fixation is not possible, there are multiple salvage techniques but with limited outcome data in this patient population. Future multicenter research on this condition will be necessary to determine optimum treatment protocols.

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