OCD Lesions of the Knee - An Updated Review on a Poorly Understood Entity

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Abstract: Osteochondritis dissecans (OCD) of the knee is a condition that has continued to perplex the orthopaedic community in its origin, despite clear advances in identification and treatment. The incidence of this potentially disabling condition has remained relatively steady, but with a shifting distribution towards young, athletic males as the primarily affected demographic. The condition commonly presents insidiously as vague knee pain but may advance to overt mechanical symptoms due to loose body formation in the joint. OCD lesions are typically classified with magnetic resonance imaging (MRI) as either stable or unstable based on the mechanical integrity of the fragment and the state of the underlying subchondral bone. The purpose of this paper is to review the current understanding of pediatric OCD of the knee, contemporary treatment principles, including methods to promote OCD lesion healing, fixation methods, and salvage techniques.

Key Points:

- High-intensity T2-signal behind the progeny fragment of the OCD lesion is suggestive of instability.
- Retroarticular or transarticular drilling of a non-healing, stable OCD lesion in the pediatric knee results in increased healing rates.
- Unstable or *ex situ* OCD lesions with intact subchondral bone can adequately be fixated using metallic screws, absorbable screws, or other biological device constructs.
- Unsalvageable OCD lesions pose a challenge to the surgeon. However, new developments with osteochondral autograft, allograft, and autologous cell techniques have increased the surgeon's ability to manage these challenging lesions.

Introduction

Much progress has been made since Franz König's first description and coining of the term osteochondritis dissecans (OCD) in 1887.[1,2] While the true pathogenesis of the condition remains incompletely understood, a great deal of knowledge has been accumulated regarding the epidemiology, anatomy, and histological components of this condition. Additionally, modern imaging techniques and surgical advances have allowed improved diagnosis and treatment. The aim of this review is to provide an updated, comprehensive review of the epidemiology, anatomy, histology, clinical presentation, and treatment of pediatric OCD of the knee.

Etiology and Epidemiology

The exact etiology of OCD of the knee is a topic of debate. Its origin is likely multifactorial, including inflammatory, vascular, traumatic, and genetic factors. The vascular theory was initially predicated on embolic causes, which relied on the assumption of an end artery epiphyseal construct.[3-5] More recent literature with advanced imaging of the epiphyseal vasculature of humans has shown similar histological changes to that seen in animals, suggesting a similar or shared pathogenesis to the development of OCD.[6,7]

There is also evidence that suggests a combination of trauma and genetics as the underlying cause of OCD lesions, as some studies have shown a connection between sporting activities and OCD lesions in up to 60% of cases.[8-10] The anatomic location of the most common solitary lesion, episodes of joint bilaterality, multiple lesions in a single joint, and twin studies support some component of genetic predisposition to the disease.[11-13] Recent evidence from Swedish researchers has identified an aggrecan gene (ACAN) that may be tied to familial OCD.[14] One potentially unifying theory involves Ribbing's theory of secondary centers of ossification, which were shown to occur in the classic OCD location on the medial femoral condyle and may be due to a nidus that later develops into an overt OCD lesion through trauma.[15]

Early epidemiological data on OCD lesions came from the Swedish registry in the 1960-1970s, which estimated peak incidences of 18/100,000 for women and 28/100,000 for men, occurring between the ages of 10-20 years old.[10] In this same study, the "classic lesion" on the lateral aspect of the medial femoral condyle was confirmed as the most common presenting location. It is important to note that this data was from a relatively small (250,000 people), homogenous white population in Malmo, Sweden. A more recent study from a larger, more diverse, regional cohort in the United States found a shifting demographic distribution. Reported incidences in this study were 9.5/100,000 for all patients and 15.4 and 3.3

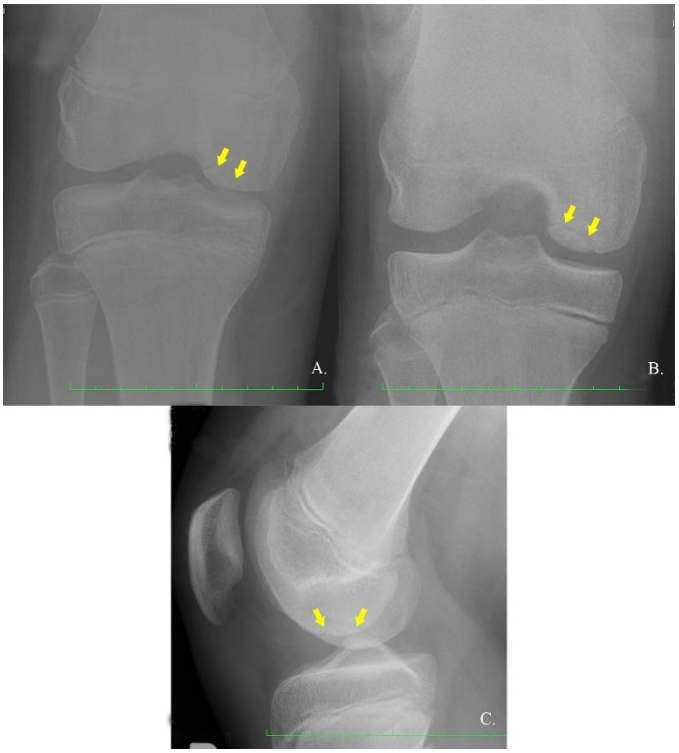

Figure 1. *Radiographs showing medial femoral condyle OCD lesion on AP (A), Tunnel (B), and Lateral (C) views (yellow arrows).*

per 100,000 for male and female patients, respectively.[16] Pareek et al. echoed a similar overall incidence in a local population-based study with 6.1 per 100,000 person-years.[17] Male sex, age between 10-20 years old, African American ethnicity, and participation in sports have all been identified as risk factors.[16,18,19]

Histology

Two important features of OCD lesions are the condition of the overlying articular cartilage and the underlying subchondral bone. Histologic study of these distinct structures has attempted to determine the etiology of OCD lesions. However, conclusive evidence as to the pathophysiology of OCD remains elusive.[20-24] A recent systematic review of published histologic studies on OCD lesions of the knee reported normal articular cartilage in some studies, degenerated or irregular articular cartilage in others, or a combination of normal and irregular articular cartilage lesions still in others.[25] In addition,

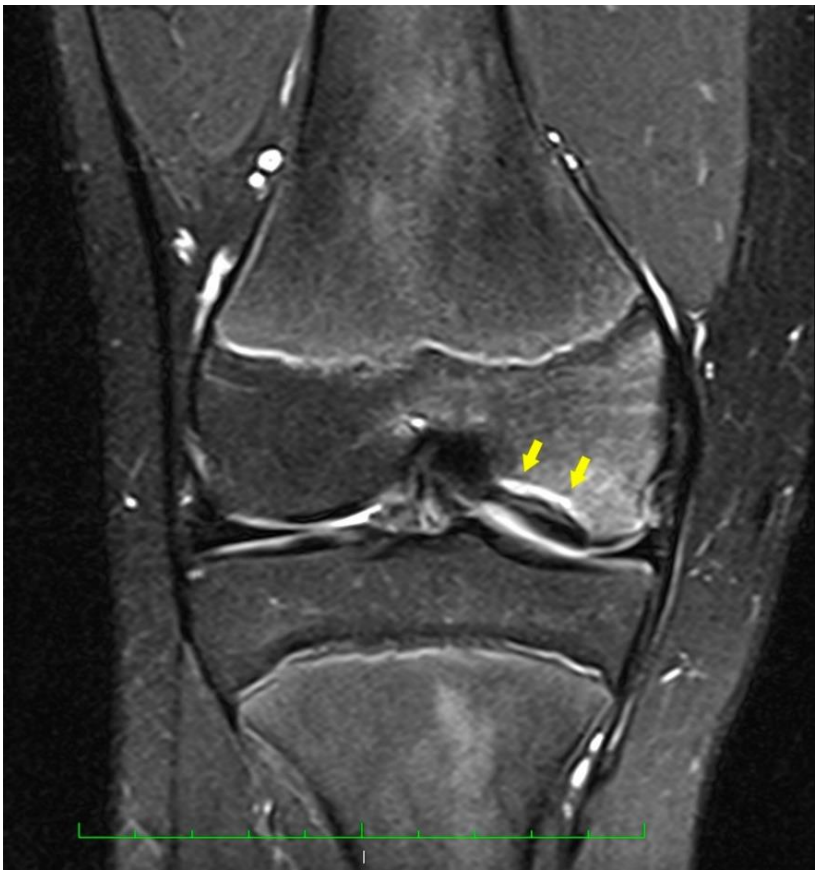

Figure 2. *Coronal T2-weighted MRI of the knee illustrating high signal intensity behind OCD lesion (yellow arrows) suggestive of instability.*

variability in subchondral bone necrosis (i.e. avascular necrosis [AVN])) was reported.[25]

A possible reason for the discrepancy in histologic findings among these studies is that OCD represents a continuum of pathology. It is likely that early-stage OCD lesions demonstrate subchondral bone viability and no evidence of AVN.[26] If AVN does occur, a defect in the subchondral bone may develop. In these cases, the defect can become filled with fibrocartilage and fibrovascular tissue at the interface between cartilage and bone and within the subchondral bone itself.[27,28] In those cases with progression, instability may result from poor structural subchondral support and the overlying articular cartilage may become diseased and unsalvageable. Understanding these processes and recognizing these histological changes helps guide the surgeon toward appropriate treatment options.

Clinical Presentation

The clinical presentation of OCD lesions depends primarily on the stability of the fragment. In stable OCD lesions, patients will often present with non-specific, generalized knee pain that is worse with higher levels of activity. These patients may also present with an effusion or altered gait, however this is not always present.[29] Tenderness may be located in the anteromedial aspect of the knee while in flexion, corresponding to the most common OCD lesion location.[30] Once a lesion becomes unstable, symptoms may progress to mechanical catching, locking, crepitus, and pain with motion. Patients may have an effusion and may also have a palpable loose body on physical exam. Depending on chronicity of the lesion, patients may also present with atrophy of the surrounding musculature. Provocative testing for OCD of the knee includes "Wilson's sign"—in this maneuver, the examiner internally rotates the tibia on the femur as the knee is actively brought from 90° of flexion into extension. The sensitivity of Wilson's sign has been shown to be poor, however.[29,31]

Imaging

Radiographs

Radiographs are an important screening test to identify the presence of OCD lesions as well as rule out other causes of knee pain. Osteochondral fractures as the result of an unstable OCD are represented on radiographs by a disruption or lucency in the subchondral bone. The ability to visualize lesions is dependent on the amount of subchondral bone that the fractured fragment contains.[32,33] Recommended radiographic views include weight-bearing anteroposterior (AP), lateral, merchant, and tunnel views (Figure 1).[11,34] Some authors also recommend

contralateral knee films, even if asymptomatic, as bilateral lesions can occur in over 20% of cases.[35,36] Full-length, lower extremity alignment views should be obtained if there is evidence of lower extremity malalignment. Jacobi et al. found an association between malalignment and the developmental location of OCD in adolescent and adult patients suggesting an increased load may contribute to the development of these lesions.[37] Radiographs are also important in the determination of skeletal maturity, which can impact management and prognosis as osteochondral lesions tend to have better healing potential in the presence of open physes.[38,39] Once the diagnosis is made, baseline radiographs can be compared to subsequent radiographs to monitor lesion progression or healing over time.[40-42]

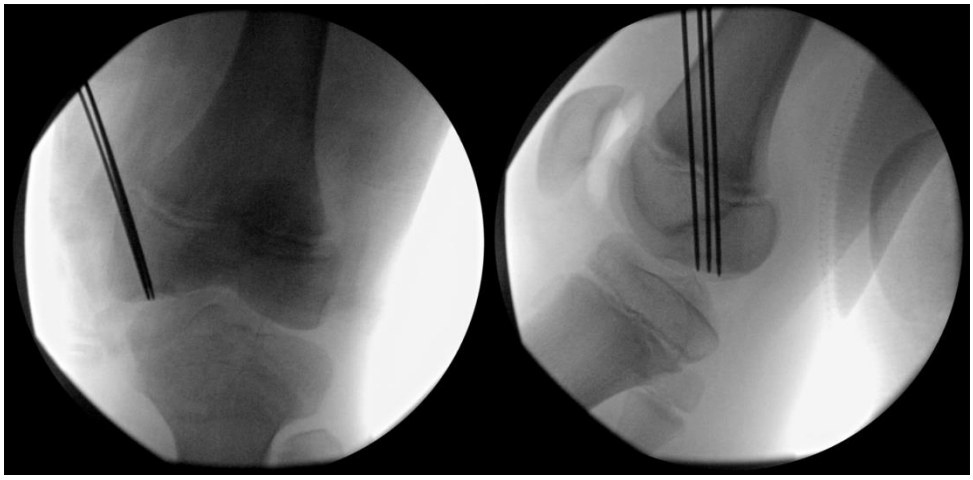

Figure 3. *Fluoroscopic images of retro-articular drilling of a stable OCD lesion in a skeletally immature patient who failed to heal with conservative treatment.*

Magnetic Resonance Imaging (MRI)

MRI is paramount in the diagnosis and characterization of OCD due to its superior soft tissue, bony architectural, and 3-dimensional imaging capabilities. The sensitivity and specificity of MRI in detecting OCD lesions has been reported to be as high as 92% and 100%, respectively.[41,43-45] MRI imaging also serves as the basis for many OCD classification systems that are widely used.[4,41,70] Although these vary slightly, they agree that identifiable loose bodies, distinct osteochondral fragments, articular cartilage disruption, and high T2 signal intensity between parent (intact) and progeny (loose) bone are all indicative of lesion instability (Figure 2). Additionally, MRI may demonstrate the presence of cartilage injuries undetected by radiographs if the bony component of the osteochondral fragment is small. MRI also allows for the detection of other structural injuries to the knee.[46]

As MRI is able to provide excellent multiplanar imaging details of the OCD lesion, its ability to detect changes in healing is far greater than x-ray. However, MRI cost and time is far greater than standard radiographs, which makes serial post-operative MRI to detect healing less ideal. Furthermore, Wall et al. have shown that the reliability of detecting healing on x-ray has been found to be substantial to excellent at 2, 4, 7, 12, and 24-month intervals.[47] Despite the excellent ability to detect healing on radiographs, some authors predict there will be a growing trend toward interval postoperative MRIs to evaluate healing.[32] Nonetheless, at this point, the American Academy of Orthopaedic Surgeons (AAOS) Clinical Practice Guidelines do not recommend for or against serial MRI imaging to evaluate healing of pediatric OCD lesions due to a lack of evidence.[48]

Treatment

The treatment for OCD lesions depends primarily on the age of the patient and fragment stability. It is widely agreed that OCD lesions in younger, skeletally immature patients have better capacity to heal compared to adults.[49] Fragment stability refers to the mechanical integrity of the lesion's underlying subchondral bone. A lesion is considered unstable if the OCD lesion is fragmented or freely mobile from the subchondral bone. The principles of OCD treatment are to promote lesion

healing and stability with the goal of preventing long-term articular cartilage injury.

Table 1. Three Phase Activity Progression for Skeletally Immature OCD Patient

	Weeks	Activity Permitted
Phase 1	1 - 6	Knee immobilization in brace or cast (depending on patient age). Patient may walk in hinged knee brace locked in extension. Brace may be unlocked for ROM 5x/day.
Phase 2	6 - 12	If the patient is pain free and radiographs show signs of healing after 6 weeks, they are allowed to begin weight bearing without immobilization and to begin a PT protocol to improve knee ROM as well as quadriceps and hamstring strengthening.
Phase 3	8 - 12	Increased activity is permitted during this phase. High impact activities and activities that involve shear stress to the knee should be restricted until the child has been pain free for several months and the radiographs show a healed lesion.

Conservative/Nonoperative

The least invasive and initial management for skeletally immature patients with stable lesions is conservative management. Nonsurgical management is also utilized for stable lesions in older patients, but the threshold to proceed to surgical treatment is lower. Although AAOS found inconclusive evidence to support any 1 particular nonoperative treatment method,[48] most would agree that activity modification with or without a period of immobilization and sports restriction for a period of 3-6 months is acceptable. Although a number of anecdotal treatment algorithms have been described, the authors' preferred treatment outline involves a 3-phase approach in skeletally immature patients has been outlined and includes progression from immobilization to graduated return to activities over a period of 12 weeks (Table 1). If properly managed, a younger patient treated conservatively has over an 80% chance of healing the OCD lesion.[50]

Transarticular and Retroarticular Drilling

In patients with stable OCD lesions who have failed to show healing with conservative treatment, transarticular or retroarticular drilling is an accepted technique with minimal reported complications. In the retroarticular approach (Figure 3), the physes are spared and studies have shown excellent results both radiographically and clinically.[51,52]

Although one advantage of retroarticular drilling is avoiding violation of the articular cartilage surface of the lesion, this approach is more technically challenging and requires fluoroscopic assistance. In contrast, a transarticular drilling technique can be done with the advantage of direct arthroscopic visualization. One disadvantage of this technique is violation of the overlying articular cartilage of the lesion. Although each drilling technique has its advantages and disadvantages, a 2013 systematic review found that both techniques resulted in acceptable radiographic healing percentages within 6 months of treatment (retroarticular drilling 86% healing at an average of 5.6 months; transarticular drilling 91% at an average of 4.5 months).[53]

Fixation

Good to excellent results using a number of fixation techniques have been reported.[54-59] Fixation of unstable OCD lesions can be achieved with a variety of implant types ranging from biological implants,[60,61] absorbable screws and implants,[62,63] and metallic implants (headless or headed metal screws).[64] Variable pitch screws have become increasingly popular for the fixation of unstable OCD lesions as they are low-profile, headless, and can provide compression of the fragment to the underlying bone (Figure 4). Most agree that a minimum of 2-3 mm of subchondral bone must be intact to achieve adequate fixation of an osteochondral progeny fragment. Careful intra-operative consideration of fixation for loose fragments should involve careful evaluation of the remaining subchondral bone on the loose, progeny fragment. Milgram et al. showed that only 50% of loose fragments had subchondral bone attached.[23]

When fixing an unstable OCD lesion, care must be taken to ensure that there is adequate purchase of the subchondral bone or the screw may back-out, causing adjacent cartilage injury on the patella or tibial plateau.[65] If a fragment is fixed arthroscopically, it is prudent to secure the screw to the screwdriver prior to placement within the joint as loss of the screw within the fat pad has been described.[65] Furthermore, care must be taken when driving the screw into the fragment, and subchondral bone as screw advancement may fracture the fragment. Removal of headed metallic screws is typically recommended once healing has occurred. This is not necessarily the case with newer screw designs, including headless screws that are fully recessed in the subchondral bone.

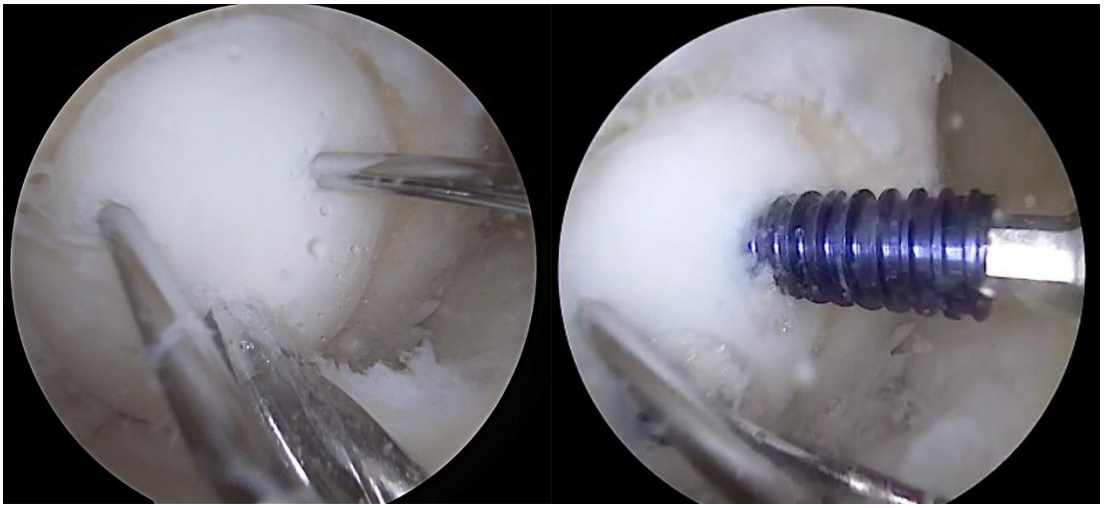

Figure 4. *Interval fixation of unstable OCD lesion with k-wire (A) and placement of cannulated variable-pitch screw for compressive fixation (B).*

To eliminate the need for screw removal, some authors advocate for fixation with bioabsorbable screws, darts, or nails.[66,67] An additional advantage of bio absorbable implants is the avoidance of metallic artifact on subsequent MRI.[66] Bioabsorbable implants are not without their complications however; absorbable screws have been shown to fail, back out, absorb asymmetrically, or even cause reactive synovitis of the joint.[68,69]

Autologous Chondrocyte Implantation

For large, unsalvageable lesions, autologous chondrocyte implantation (ACI) presents a viable option, albeit one that involves a staged intervention. The third-generation matrix-induced autologous chondrocyte implantation (MACI–Varicel Pharmaceuticals Inc, Cambridge, MA) is the first product approved by the FDA that employs tissue engineering to grow autologous articular cartilage cells on a scaffold. Although not currently established as safe or effective by the FDA in pediatric patients, this procedure has been performed in patients under the age of 18 in other countries.[70,71] Relative contraindications for ACI include bovine allergy since the chondrocytes are cultured in bovine serum, which may potentially limit patient cell growth if an allergy exists.

During the first stage of ACI/MACI chondrocyte harvest, it is important to address any associated intra-articular pathologies encountered (e.g. meniscal injuries, ligamentous injury, loose bodies, etc.) It is important to recognize that if there is a bipolar lesion (e.g. patella defect with an articulating trochlear lesion) or an uncontained defect with > 8 mm subchondral bone loss, that ACI/MACI is contraindicated and other techniques may need to be implemented (Figure 5).[72]

Osteochondral Autograft Transplantation

Osteochondral autograft transplantation (OATS) is commonly used for small to medium-sized (0.5–3 cm^2) symptomatic lesions that have associated subchondral bone loss.[73] One must first debride the damaged site and determine the proper size and contour of the needed autograft. The autograft harvest site is from a non-weight bearing portion of the knee (typically, the lateral

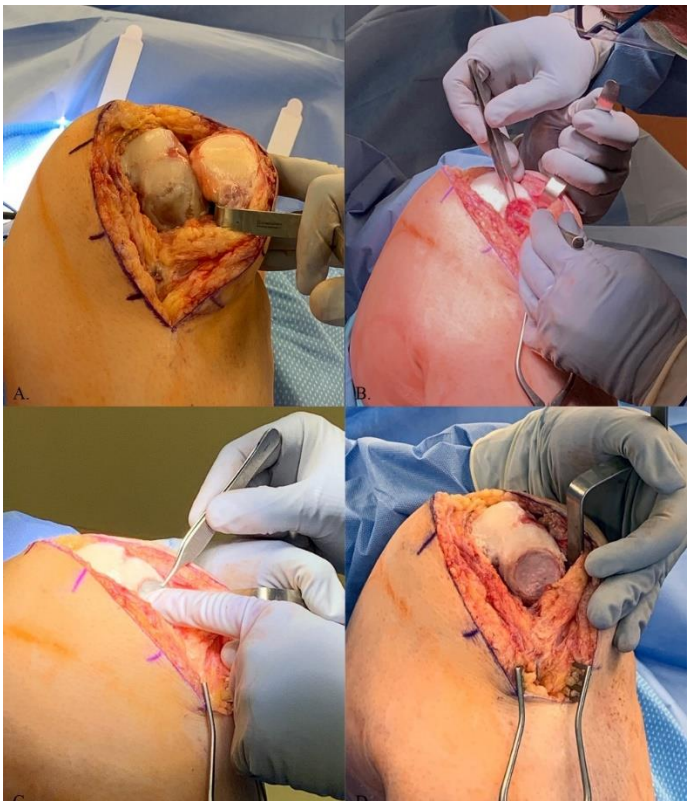

Figure 5. *Large OCD lesion with subchondral bone loss (A) subchondral autograft placement after debridement (B) placement of MACI biologic patch (C) and view of MACI patch after final preparation and placement.*

trochlea), and the graft is press-fit into the lesion. During the procedure, at least 10 mm of subchondral bone should be harvested. Grafts that are congruent or countersunk up to 1 mm fare well, however those that are countersunk > 1 mm or that remain proud have been shown to have higher failure rates.[74,75] If multiple plugs are required to fill a larger defect, a "mosaicplasty" can be performed, however, this procedure appears to have less favorable outcomes when compared to ACI.[76]

Drawbacks to the OATS procedure for OCD lesions include potential donor site morbidity from the harvest site, difficulty in matching the exact defect shape, and the limited amount of donor site cartilage that can be harvested, particularly in pediatric patients where the physis can limit options for harvest[77].

Osteochondral Allograft Transplantation

Osteochondral allograft transplantation is a widely accepted treatment for large defects ≥3 cm^2.[78] This technique has also been shown to be an effective salvage procedure in cases of failed cartilage repair, with studies citing upwards of 78%-90% graft survival rates and improved subjective knee scores at 10 years.[79-81] Drawbacks of the procedure are based primarily on cost and availability of matched donors, in addition, to the potential for disease transmission.[73,82]

Regarding athletes and return to play, some case series have shown success, with over 90% of patients in one study being satisfied with the procedure and 79% of patients returning to high- level activity.[83] Positive results have been shown in the femoral trochlea, with survival rates of 87.9% and 77.2% at 5 and 10 years, respectively, in a recent systematic review. Revision surgery, patellar, and bipolar lesions have been shown to have worse outcomes overall than other lesions.[81,84,85]

Conclusion

Young patients presenting to a pediatric or sports medicine clinic with OCD lesions are not an uncommon finding. While no definitive etiology has been established for OCD, multiple factors may contribute to the development of the lesion, including mechanical, vascular, and genetic factors. When approaching OCD lesions, proper imaging starting with radiographs and a thorough physical exam is paramount. After a trial of non-operative management, further imaging such as MRI is critical in evaluating lesion characteristics to determine which of the many treatment options available is appropriate for the particular patient. Treatment should be tailored to the characteristics of the patient and the lesion. A stable lesion should first be treated with conservatively, especially in those with significant growth remaining. If a stable lesion fails to heal, retroarticular or transarticular drilling is an excellent option for those that are skeletally immature. For salvageable unstable lesions, numerous fixation techniques can be used. If the OCD lesion is unstable

and unsalvageable, options such as MACI, osteochondral autograft, and osteochondral allograft remain available for treatment.

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