

45 Osteochondritis Dissecans

Cecilia Pascual-Garrido, Taylor M. Southworth, Mark A. Slabaugh, Neal B. Naveen, Nicole A. Friel, Ben U. Nwachukwu, and Brian J. Cole

INTRODUCTION

Osteochondritis dissecans (OCD) results in the destruction of subchondral bone with secondary damage to overlying articular cartilage (1,2). The prevalence of this condition is estimated to be between 11.5 and 21 per 100,000 (3,4). OCD is classically divided into juvenile and adult forms based on the patient's skeletal maturity (1). Juvenile OCD (JOCD) lesions occur in children and young adolescents with open growth plates. Although adult OCD lesions may arise de novo, they more commonly result from an incompletely healed and previously asymptomatic JOCD lesion (5). JOCD lesions have a better prognosis and higher rates of spontaneous healing with conservative treatment than do adult OCD lesions (6). Adult OCD lesions have a greater propensity for instability and, once symptomatic, typically follow a clinical course that is progressive and unremitting (7). While lesions most frequently occur on the femoral condyles, they are also found in the elbow, wrist, ankle, and femoral head (8–12). The highest incidence rates in JOCD are among patients ages 10 and 15 years old, ranking among the most common causes of knee pain and dysfunction in young adults (6,7,13) (Fig. 45-1).

Fig. 45-1

The typical presentation of OCD in the knee includes pain and swelling related to activity. Instability is not usually reported, though mechanical symptoms, such as catching and locking, usually occur in the presence of a loose body and are frequently the initial presenting symptoms. The patient may walk with an antalgic gait or with the leg externally rotated to decrease pressure over the lesion, known as Wilson sign (14). Effusion, decreased range of motion, and quadriceps atrophy are variably present depending upon the severity and duration of the lesion (15). Patients typically have tenderness localized over the lesion. More than 70% of OCD lesions are found in the lateral aspect of the medial femoral condyle (MFC), intersecting the intercondylar notch near the posterior cruciate ligament (PCL). Central lateral condylar lesions account for 15% to 20% and femoral trochlear lesions for <1%. Patellar involvement is uncommon (5%–10%) and, if present, is typically located in the inferior medial area (7).

INDICATIONS

Operative treatment for OCD lesions is focused on improving the blood supply to the lesion, restoring the joint surface, and providing rigid fixation (16). Surgery is indicated both for young patients with detached or unstable lesions as well as for those approaching physeal closure whose lesions have been unresponsive to nonoperative management (1,17). Operative treatment may also be considered in patients who are unable to participate in a prolonged nonoperative course.

Specifically, interventions such as drilling or internal fixation are indicated for the symptomatic juvenile patient who has failed a course of 3 to 6 months of nonoperative treatment. However, when the presence of significant mechanical symptoms dominates the clinical presentation, the decision to operate might occur earlier. Drilling is generally limited to young patients with open physes and low-grade lesions, Guhl grades I and II, respectively, that are not grossly unstable with palpation.

Higher grade OCD lesions with articular cartilage flaps or loose bodies, Guhl grades III and IV, respectively, are generally not amenable to conservative treatment. Ananthaharan and Randsborg

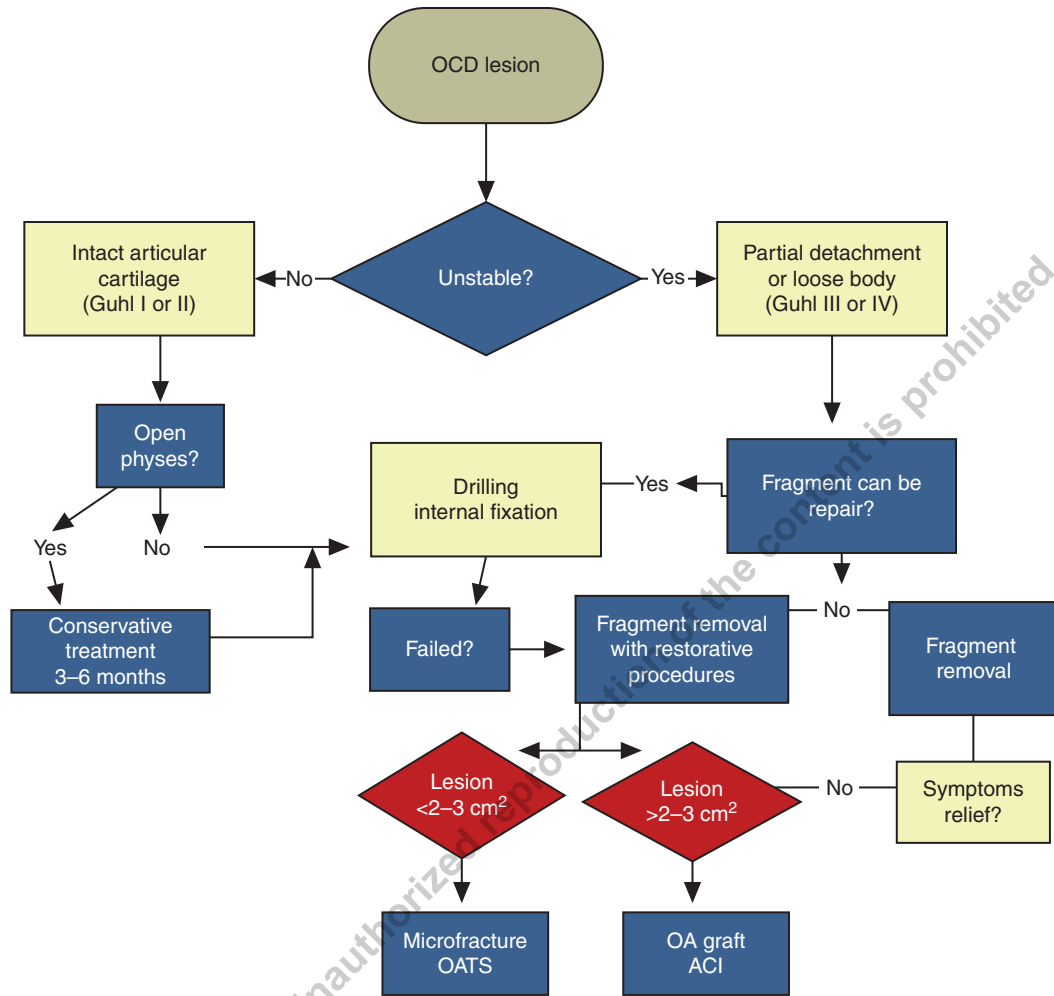


FIGURE 45-1

Algorithm for surgical treatment of JOCD/OCD. Surgical goals should always try to reestablish the joint surface and conserve the osteochondral fragment. If not, restorative treatment should be implemented.

found that patients with grade III and IV lesions were five times more likely to fail conservative treatment when compared to those with grades I and II lesions (3). Thus, fixation of partially detached lesions or loose bodies is appropriate for large fragments containing sufficient subchondral bone to provide union and support of the fixation system. Lower grade lesions, Guhl grades I or II, may also be fixed if conservative treatment has failed or there is clinical suspicion of instability. Additionally, unstable “trap door” lesions that are partially elevated from the subchondral bed require fixation (18,19).

Many patients function well despite having OCD lesions and only become symptomatic when the OCD fragment becomes unstable. In these cases, fragment removal is indicated and can lead to symptomatic relief. For example, if the defect is relatively small, from an area with less contact pressure (20), such as the “classic” OCD lesion located near the PCL origin on the “upslope” of the MFC, and is associated with the acute onset of mechanical symptoms in a skeletally mature adult, fragment removal and observation are indicated. In contrast, complaints of “achy discomfort,” effusions unrelated to mechanical symptoms, and weight-bearing pain over the lesion may be indicative of symptoms that are due to the defect itself rather than the unstable or displaced fragment. These lesions are an indication for a cartilage restoration procedure, such as a marrow stimulation technique, osteochondral autograft, osteochondral allograft (OCA), or autologous chondrocyte implantation (ACI). Specifically, microfracture is indicated in patients with a localized cartilage defect (<2–4 cm²). Patients with low demand

and bigger lesions can also improve with this technique. The indications and optimal patient population for osteochondral autograft remain narrow. A single plug autograft is preferred for defects smaller than 1 cm². However, some authors perform mosaicplasty with multiple smaller plugs on defects as large as 4 cm² with encouraging results (21). Larger OCD lesions (>2 cm²) may be treated with OCA transplantation (22). OCA transplantation provides the ability to resurface larger and deeper defects with mature hyaline cartilage, while addressing the underlying bone deficiency. ACI is ideal for symptomatic, unipolar, well-contained chondral and osteochondral defects measuring between 2 and 10 cm² with bone loss <6 to 8 mm (Fig. 45-1).

In contrast to JOCD where nonoperative treatment is usually first line, adult OCD typically requires early surgical intervention (5). Additionally, in cases of chondral separation, surgical results are better than those with nonsurgical treatments (23,24).

CONTRAINDICATIONS

Surgical decision-making is based on patient age, skeletal maturity, lesion appearance, and clinical symptoms. The ideal goal of conservative treatment is to obtain lesion healing before physal closure. Stable OCD lesions in young patients have a favorable prognosis when treated initially with nonoperative treatment. A large multicenter review of the European Pediatric Orthopedic Society study (509 knees, 318 juvenile, and 191 adult in 452 patients) suggests an improved prognosis with conservative treatment in young patients with a small lesion (<2 cm²) in the classic location with no signs of dissection or effusion (23). Ananthaharan and Randsborg noted that 78% of grade I and II lesions were successfully treated with conservative management (3). Moreover, spontaneous healing of JOCD lesions has been reported when the lesion is not in the classical location of the lateral aspect of the MFC (25).

Traditional nonoperative treatment for JOCD consists of an initial phase of knee immobilization with partial weight bearing (4–6 weeks) in those patients in whom no detachment is noted on MRI. Once the patient is pain free, weight bearing as tolerated is permitted and a rehabilitation program emphasizing knee range of motion and low-impact strengthening exercises ensues. If there are radiographic and clinical signs of healing at 3 or 4 months after the initial diagnosis, patients may participate in a gradual return to sports with increasing intensity allowed in the absence of knee symptoms. The likelihood that the lesion will heal with this management is approximately 50% at 10 to 18 months (9).

Q1

Neither the literature nor our experience allows us to definitively determine whether untreated OCD, either with the fragment in situ or following fragment excision, has a more significant likelihood of developing into symptomatic degenerative joint disease in the future. For example, Linden performed a long-term retrospective follow-up study of patients with OCD of the femoral condyles with an average follow-up of 33 years after initial diagnosis (13). It was concluded that while individuals who are older when the osteochondritis manifests, such as those with adult OCD, have an increasing incidence of knee osteoarthritis (OA), JOCD is not associated with an increased risk of OA when compared to the normal population. In contrast, Twyman et al. completed a prospective follow-up of 22 knees with JOCD into middle age and found that 50% had some radiographic signs of OA (26). The likelihood of OA development was found to be proportional to the size of the area involved.

PREOPERATIVE PREPARATION

Standard anteroposterior and lateral radiographs of the knee permit localization of the lesion and assessment of the physal status of the patient. Additional images such as tunnel or sunrise views are useful for suspected distal MFC or patellar lesions, respectively. By convention, lesions may be anatomically localized using the Cahill classification (1) (Fig. 45-2). Magnetic resonance imaging (MRI) is the mainstay in diagnosis of OCD lesions. Lesion qualities including bone edema, subchondral separation, and cartilage condition may be evaluated prior to determining a treatment course (Fig. 45-3). Intraoperatively, OCD lesions may be classified using the criteria suggested by Guhl (27) (Table 45-1).

Table 45-1

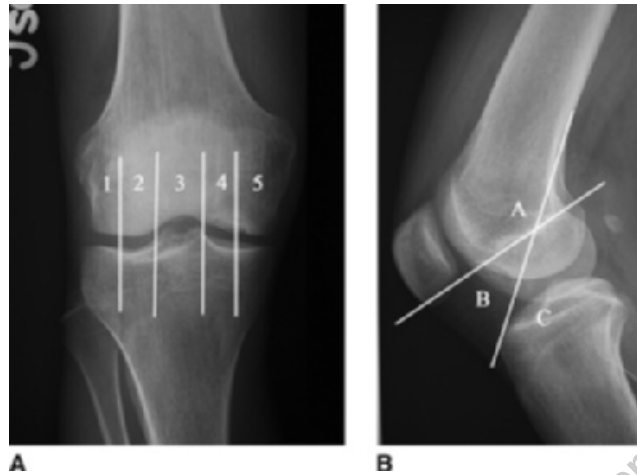


FIGURE 45-2

A: Anteroposterior radiograph of a 35-year-old male with an OCD lesion occupying the weight-bearing area of the femoral condyle. As per Cahill's classification, numbering of the five anatomic areas begins in the middle side. The condyles are bisected, and area 3 is bounded by the walls of the intercondylar notch. **B:** Lateral radiograph of the same OCD lesion. The line separating A and B represents the roof of the intercondylar notch. The line separating B and C is a continuation of the posterior femoral cortex.

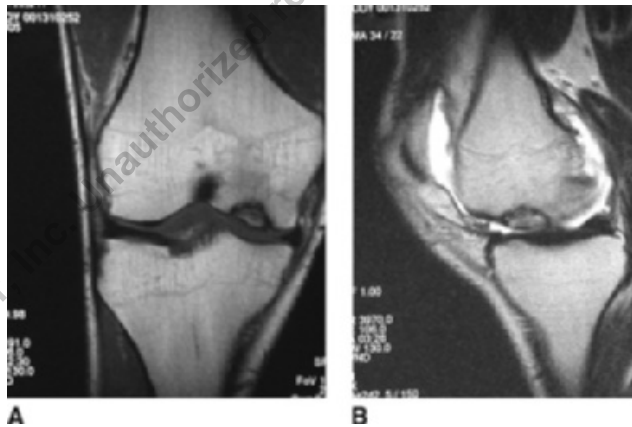


FIGURE 45-3

A: Coronal MRI of the knee of an OCD lesion of the MFC of the left knee. Note the low-intensity signal between the osteochondral fragment and the subchondral bone, suggesting an unstable fragment. **B:** Sagittal MRI through the MFC.

TABLE 45-1 Description of the Guhl Classification

| Grade | Intraoperative Finding |
|-------|---|
| I | Normal articular cartilage |
| II | Fragmentation in situ |
| III | Partial detachment |
| IV | Complete detachment, loose body present |

Source: Guhl JF. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res.* 1982;167:65–74.

OCD LESION TREATMENT TECHNIQUES

Reparative Treatments

The goal of reparative procedures is to restore the integrity of the native subchondral interface and preserve the overlying articular cartilage (28).

Drilling

The disruption of subchondral blood supply is thought to be an important factor in the development of OCD (29). Drilling involves the creation of vascular channels to the devitalized region in order to restore blood flow and enhance healing.

Antegrade drilling involves drilling through the articular surface and into the femoral epiphysis. It is done arthroscopically under direct visualization (30–32). If the lesion is not accessible via standard portals, accessory portals are created to obtain an orthogonal drilling angle. A K-wire 2 cm longer than a small cannula facilitates the direction and depth of the channels (29). Return of blood and fat droplets through the articular surface confirms penetration of cancellous bone.

More commonly, drilling is performed by entering at a nonarticular location. For example, the classic OCD lesion is located along the lateral aspect of the MFC and can be accessed at the anterior aspect of the PCL origin along the inner margin of the MFC with a K-wire introduced percutaneously or through the inferolateral portal.

Retrograde drilling is inherently more difficult when targeting the lesion base. C-arm visualization is needed to help avoid joint penetration or dislodgement of the OCD fragment. Alternative methods, including sonography (33) and the use of an ACL guide (34), have been proposed. Large-diameter drilling with iliac crest bone graft supplementation has also been described (35).

Internal Fixation

Fixation can be accomplished with bone pegs, osteochondral grafts, or metal or bioabsorbable devices (29,36,37). As mentioned previously, unstable “trap door” lesions that are partially elevated from the subchondral bed require fixation (11,19). If accessible, the base of the lesion and bony surface of the flap are debrided. Microfracture awls can be used to penetrate the base and allow improved access to the subchondral blood supply. The fragment is reduced and temporarily fixed with K-wires to facilitate the final placement of the fixation device. In most cases, fixation is accomplished at two or more locations to impart compression and rotational stability to the fragment. This is important as *in vitro* studies suggest that compression, resulting in friction between the fragment and the base, improves stability and resistance to shear loading. All devices should be recessed beneath the cartilage surface, with metal screws being removed postoperatively when evidence of union is seen (Fig. 45-4), typically 6 to 8 weeks later. Hardware removal allows for second-look arthroscopy, which provides the benefit of verifying defect healing. Additionally, hardware removal is an added plus as the screws may become prominent and bothersome should the fragment settle around the fixation device. Bioabsorbable fixation is also an option, especially when patients desire

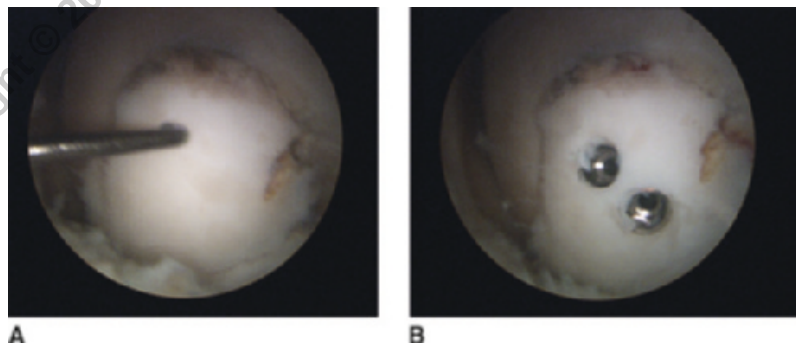


FIGURE 45-4

A: Intraoperative, arthroscopic view of OCD lesion of the MFC in a 20-year-old male. **B:** Anatomic reduction and fixation of the osteochondral fragment was performed with compression screw fixation.

to avoid a second surgery. Bioabsorbable screws are often made from biphasic calcium phosphate and poly-L,D-lactic acid or amorphous poly-L-lactic acid, which allows for controlled degradation over time.

Restorative Treatments

Restorative procedures attempt to replace damaged cartilage with hyaline or hyaline-like tissue (30,38,39). In the event either that the OCD fragment cannot be initially stabilized and requires excision or that the fragment fails to heal after initial fixation, it is important to determine the clinical relevance of the remaining defect to decide if a cartilage restoration procedure is the best next step. Most importantly, identifying and treating each patient with relevant comorbidities such as malalignment and meniscal and ligament deficiency is imperative to render successful treatment.

Marrow Stimulation Techniques

Abrasion chondroplasty, subchondral drilling, and 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 (40). The calcified cartilage layer is carefully debrided, and surgical awls are used to penetrate the subchondral bone to enhance defect fill (41) (Fig. 45-5).

Osteochondral Autograft Transplantation

Osteochondral autograft transplantation involves transplanting osteochondral tissue from a non-weight-bearing region of the patient's own knee to restore the articular surface. The technique has been well described (27) and includes the careful consideration of minimizing donor-site morbidity during graft harvest. When treating the defect with an osteochondral autograft, specific attention should be paid to recreating the natural contour of the condyle with accurate depth and plug placement to avoid graft instability due to relative noncontainment. These steps are critical to avoid early failure.

Osteochondral Allograft Transplantation

Osteochondral allograft transplantation, in contrast to autograft, involves transplanting osteochondral tissue from a donor cadaver. From the donor, an osteochondral plug is able to be harvested. Commercially available instrumentation systems permit accurate sizing and matching of a cylindrical allograft plug to the defect. To do this, a 2.4-mm guide pin is driven through the cannulated sizing guide into the base of the defect. The guide is then removed and used to size the allograft at the appropriate topographic location on the donor. A cannulated cutting reamer of the same size is used to ream to a depth of approximately 6 to 8 mm. Usually, it is possible to press fit the graft with the use of an oversized tamp to secure it. However, if necessary, fixation of the allograft with bioabsorbable compression screws or headless differentially pitched titanium screws is performed.

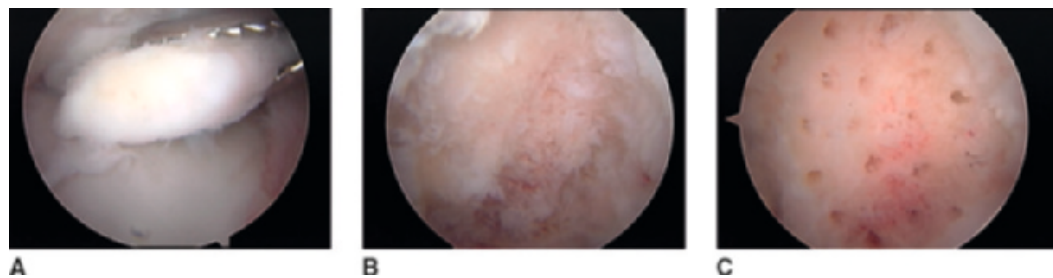


FIGURE 45-5

A: Intraoperative, arthroscopic view of an unstable osteochondral fragment. **B:** Removal of the unstable lesion, revealing the underlying subchondral bone. **C:** Microfracture holes throughout the entire area of the OCD lesion.

Autologous Chondrocyte Implantation

In ACI, healthy chondrocytes are biopsied from a non-weight-bearing region and expanded in vitro over 4 to 6 weeks. Alternatively, the cells may be cryopreserved for up to 5 years and utilized when necessary. At implantation, defect preparation involves debriding to the calcified cartilage base and creating vertical walls of healthy cartilage to shoulder the lesion. A periosteal patch from the proximal tibia or synthetic collagen membrane is attached to the perimeter using interrupted 5-0 or 6-0 Vicryl sutures. The edges of the patch are sealed with fibrin glue and the cells injected beneath the patch into the virtual chamber. In third-generation ACI, or matrix-induced ACI (MACI), the chondrocytes are added to a collagen membrane prior to implantation, thus removing the need for a periosteal patch or injection of cells.

Defects deeper than 8 to 10 mm can be approached by concomitant or staged bone grafting. Bone grafting should be performed up to the level of the subchondral bone (42). Prior to bone grafting, drilling through the bed following debridement allows appropriate blood flow into the defect, ensuring subsequent bone graft incorporation. When bone grafting is performed as a primary procedure in an effort to stage for definitive treatment with ACI, most surgeons will wait for a minimum of 6 months to allow for bone graft incorporation. Limited experience exists with anecdotal results of a “sandwich” technique where bone grafting is performed in combination with ACI using a “periosteal sandwich” with both cambium layers facing one another and the cells injected in between.

PEARLS AND PITFALLS

Important pearls and pitfalls involve the clinical decision of the appropriate disease management.

- If a too advanced procedure is performed too early, it limits the options for future procedures.
- Nonoperative course should be first-line treatment in the majority of cases.
- Detached or unstable lesions require surgical intervention.
- Adult OCD lesions often require early surgical intervention.
- Guhl grades III and IV do not respond well to conservative management.
- Be careful to differentiate between symptoms caused by the fragment versus symptoms caused by the defect to determine if a cartilage restoration procedure is needed.

POSTOPERATIVE MANAGEMENT

Authors who focus on the biology of the fragment–subchondral bone interface argue that the knee should be protected in a knee immobilizer and treated similar to an intra-articular fracture (1). Alternatively, some authors place a premium on the health of the articular cartilage and note the value of continuous motion (1). Hughston et al. demonstrated the detrimental effects of prolonged immobilization, including stiffness, atrophy, osteopenia, and, potentially, chondropenia (4).

In the senior author’s practice, after internal fixation patients may heel-touch weight bear and utilize continuous passive motion (CPM) machines for 4 to 6 hours per day. For postoperative management after a marrow stimulation procedure, instructions depend on the location of the lesion. For microfracture of the femoral condyle, no brace is usually required, but a CPM machine is recommended for 6 to 8 hours per day. Patients begin initially on crutches with 20% to 30% weight bearing. For microfracture of the patella or trochlea, patients are instructed to wear a brace locked to achieve up to 40 degrees of flexion until the first postoperative visit. These patients also are able to weight bear as tolerated and are recommended to use CPM machines for 6 hours per day. After osteochondral autograft transplantation, protected weight bearing is encouraged for up to 6 weeks postoperatively. For OCAs, restricted weight bearing is recommended for at least 8 weeks. The senior author’s preferred postoperative management program for OCA has been previously described (43). Patients use a hinged-brace locked in extension for 2 weeks followed by an additional use of 2 to 4 weeks. During this time, the patient is restricted to touch-down weight-bearing protocol. The brace is discontinued after the patient is able to demonstrate a straight leg raise without extension lag. Partial weight bearing begins at 6 weeks post-operatively and is gradually progressed to full weight bearing. Patients are able to begin higher impact activities beginning at 6 to 8 months. For ACI postoperative management, similar to many other procedures, non-weight bearing and CPM is indicated.

Q3

Complications

Complications, like indications, are both general and specific to each surgical technique. General complications include technique failure or failure to relieve patients' symptoms requiring an additional reoperation. Interestingly, Kramer et al. found that female sex, prolonged duration of symptoms, and internal fixation may be associated with worse outcomes in terms of residual pain, rates of reoperation, and return to sport. Kramer et al. also found that of 26 patients who underwent transarticular drilling, drilling with fixation, or a marrow stimulation procedure with excision, 14% required unplanned reoperation (44). Complications associated with OCD fixation include damage to opposing cartilage surfaces from proud hardware, broken hardware, loose bodies, and synovitis (24,45,46). Osteochondral autograft transplantation complications include donor site morbidity, which is why lesion size is one of the limiting factors in using this technique. Potential complications of OCAs include poor graft contour matching, immunogenicity, and possible disease transmission, the risk for the latter two being exceedingly uncommon.

Results

Early studies evaluating the results of treatment of these lesions focused on fragment excision. Denoncourt et al. treated 37 patients with arthroscopic removal of the fragment and curettage of the lesion (47). They reported complete "healing" in 10 cases by second-look arthroscopy. They recommended this treatment in adults and children who have failed initial attempts at nonoperative treatment. Similarly, Ewing and Voto excised the fragments and drilled the defect in 29 patients (17). They reported a satisfactory result in 72% of their patients with short-term (<1 year) follow-up. Recent reports suggest that fragment excision may provide short-term pain relief but not provide long-term success with further follow-up. Anderson et al. evaluated 19 patients with 20 OCD lesions who were treated with fragment excision (29). Follow-up between 2 and 20 years showed that only five patients could participate in strenuous activity without significant symptoms. Eleven patients had pain with activities of daily living, and the remaining three patients had pain with light activities. It was concluded that fragment excision may show improvement of the symptoms in the short term, but the remaining defect and involved compartment may worsen with time.

Outcomes of OCD drilling are generally favorable, with patient age being the best prognostic factor. Individuals with OCD diagnosed and treated with drilling as an adult have decreased radiographic healing and less favorable symptom outcomes (48). Louisia et al. noted 71% (12/17) radiographic healing and two poor results in JOCD compared with 25% (2/8) healing and four poor results in adult OCD patients (32). Overall, good to excellent results are observed in >80% of adolescent patients, with 70% or more being able to return to sports (7,8,32,34). It is our opinion that the ideal patient with symptomatic OCD to treat with drilling is when the defect is grossly stable to palpation despite some MRI evidence of fluid behind the fragment indicating biologic instability. Occasionally, we will augment the treatment of these lesions with the placement of a bioabsorbable differentially pitched threaded compression screw (Arthrex, Inc., Naples, FL).

Favorable outcomes after internal fixation of OCD fragments have been reported for both metallic and bioabsorbable devices. Kivistö et al. noted good to excellent results in 86% of young patients treated with staple fixation (53% radiographic healing) (46). A study of Herbert compression screw fixation yielded 13/15 (87%) normal knees by IKDC grading and radiographic healing in 93% (49). Gomoll et al. evaluated 12 adolescent patients with unstable Cahill Type 2C lesions treated with compression screw fixation with average 6-year follow-up (36). All lesions healed without clinical or radiographic evidence of degenerative disease. Fixation with self-reinforced poly-L-lactic acid nails and pins permits radiographic healing in 60% to 100% of cases (50,51). A cohort study by Weckström et al. suggests that implant geometry (i.e., presence of barbs or a flared head) is a factor in successful outcomes (52).

Ishikawa et al. studied 13 knees in 13 patients with open physes who were treated with internal fixation with an average follow-up of 22.92 \pm 10.95 months. The study found that 76.9% patients significantly improved in Lysholm score postoperatively. Twenty-three percent of these patients required revision surgery, all of which were considered Guhl grade II arthroscopically but grade III on MRI on the grading system described by Dipaola (53). Kubota et al. evaluated

22 patients who underwent internal fixation for OCD fragments, 14 of which were skeletally mature, and found Lysholm score was significantly improved from baseline at both short-term (35.4 months) and mid–long-term (142.5 months) follow-up, although it was also significantly better at short-term follow-up when compared to mid–long-term follow-up. In contrast, Tegner activity scale was deteriorated at mid–long-term follow-up compared to preoperative scores and short-term scores. The authors noted this was likely due to patients discontinuing sports due to age rather than knee symptoms (54). In a retrospective cohort study, Wu et al. studied 87 patients with unstable lesions who underwent internal fixation for a mean follow-up of 60 months and found that 76% of the lesions showed healing beyond 2 years postoperatively. The study noted 24% of these fixations either failed but found no difference in failure rates between patients with open versus closed physes (55).

Q4

Chadli et al. evaluated nine patients with either ICRS grade II or III lesions who were treated with hybrid fixation, which involves the addition of a biologic fixation to the mechanical fixation, such as an osteochondral graft, with a median follow-up of 10.1 years (7–14 years). While this study did not compare preoperative and postoperative outcomes, it did evaluate the incorporation of the graft during hardware removal and noted the plugs were integrated but with a superficial peripheral chondral gap (56).

Gudas et al. suggested that OCD lesions treated with microfracture have a significantly worse clinical outcome than do traumatic cartilage lesions (57). Normally, large lesions (more than 2 cm²) treated with microfracture demonstrate deterioration with time due to decreased fibrocartilage resilience and stiffness (58). Knutsen et al. compared results at 2- and 5-year follow-up in 80 patients who had a single chronic symptomatic cartilage defect on the femoral condyle of the knee, treated randomly with microfracture or ACI (59). Twenty-eight percent of these lesions were due to OCD. Both treated groups showed satisfactory results in 77% of the patients at 5 years. No significant difference between the two treatments groups was evident. They proposed that microfracture should be preferred as first-line treatment option for defects located on the medial or lateral femoral condyle of the knee. Microfracture should therefore be considered as the first-line treatment in lesions <2 cm² with subchondral bone integrity and in patients with lower physical demand levels and slightly larger lesions (2–4 cm²).

The advantages of the osteochondral autograft transplantation technique include absence of disease transmission risk and the lower cost of a single-stage procedure. Disadvantages include donor site morbidity and limited available graft volume. In addition, it is technically difficult to position the plugs to recreate the exact contour of the condylar surfaces. Despite these limitations, results from isolated small to medium-sized lesions of the femoral condyle have been good: 91% good to excellent results at >3 years (21). Interestingly, Miniaci et al. have suggested using the OATS technique for the fixation of unstable OCD lesions of the knee. Twenty patients with OCD lesions were fixed in situ by using multiple 4.5-mm osteochondral dowel grafts harvested from the edges of the trochlea. At 18 months postoperatively, all knees were scored as normal and radiographically healed at 6 months postoperatively (37). Advantages of this technique include the fact that a considerable volume of the original lesion is replaced by autologous bone graft. This technique was used to provide stable biologic fixation using autogenous bone graft. Outerbridge et al. reported favorable short-term results using autografts harvested from the lateral facet of the patella in 10 patients with large femoral OCD lesions (60).

In a cohort of 64 patients treated with fresh OATS, 72% had good to excellent clinical outcomes at 7.7 years after surgery (61). Garret et al. reported on a series of 17 patients treated with a OAs with 94% clinical success at a mean follow-up of 3 years (5). McCulloch et al. studied the clinical outcomes on 25 patients who underwent prolonged fresh OA (these grafts are harvested and are typically maintained refrigerated at 4°C for up to 28 days). Six of these patients were diagnosed with OCD. They reported 84% patient satisfaction and 88% radiographic incorporation of prolonged fresh allografts to the femoral condyle (58). Convery et al. reviewed retrospectively 12 patients treated with OA grafts with a mean follow-up of 5 years. Four of these patients had OCD. Overall, outcomes were rated as excellent in all but one patient, who had a gross technical deficiency. The need for technical proficiency in performing fresh osteochondral allografting was assessed (62). In summary, treatment of OCD with osteochondral allografting provides subjective improvement in 75% to 85% of patients and has the longest-term follow-up in the literature (36) (Fig. 45-6).

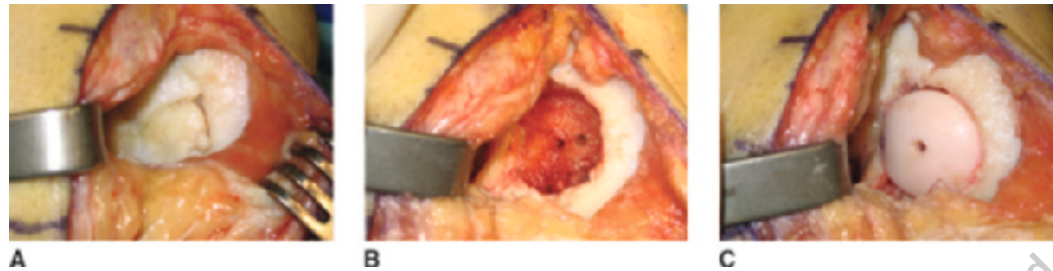


FIGURE 45-6

A: Intraoperative photo demonstrating a large OCD lesion located on the MFC. **B:** The OCD fragment was removed, and the recipient site was cored. **C:** The lesion was treated with a fresh OA.

In a case series, Cotter et al. studied 37 skeletally mature patients (39 knees) who underwent OCA after failure of initial OCD treatment with an average follow-up of 7.29 \pm 3.30 years. The study found significant improvement in all patient-reported outcome scores, except the Short Form 12 mental subscale. Of note, 81.8% of these patients returned to sport at an average of 14.0 \pm 8.7 months; 35.9% of patients underwent subsequent surgery, and 5% reported OCA failure (43).

In another case series, Sadr et al. followed 135 patients, 149 knees, with OCD treated with OCA with a median follow-up of 6.3 years (1.9–16.8 years). These patients displayed significant improvement in pain and function scores, and 95% stated they were satisfied with the procedure. Of the 149 OCAs performed, 23% underwent reoperation and 8% were classified as failures. Graft survivorship was noted to be 93% at 10-year follow-up (63). Along with Cotter et al., these studies show that although there is a relatively high reoperation rate, failure rates are low when OCA is used as treatment for OCD.

Peterson et al. evaluated 58 patients (mean age: 26.4) with OCD who underwent ACI after a mean follow-up of 2 to 10 years. Thirty-five patients had JOCD and 23 adult OCD. Integrated nonarticular cartilage repair tissue had formed (46), and successful clinical results were noted in more than 90% of patients. Only 30% of the 27 patients with preoperative and postoperative radiographs showed joint space narrowing. ACI appears to be a reasonable alternative in OCD lesions (64). Results evaluated at a minimum 2-year follow-up essentially mirror that reported in the literature: 76% successful outcomes at 4-year follow-up (65) (Fig. 45-7).

SUMMARY

OCD of the knee requires a timely diagnosis in order to prevent compromise to the articular cartilage and maximize opportunity to perform a restorative procedure. Indications for surgical treatment are based on lesion stability, skeletal maturity, and clinical symptoms. Reestablishing the joint surface, improving the blood supply of the fragment, rigid fixation, and early motion are primary goals for osteochondral fragment preservation. When the fragment is not suitable for preservation, careful consideration of defect location and the patient's clinical presentation will determine when cartilage restoration procedures should be utilized. Successful restorative options should relieve pain, restore function, and prevent the development of secondary OA.



FIGURE 45-7

A: Lesion of the lateral femoral condyle, circled to define the borders for ACI. **B:** Preparation of the defect. **C:** Lesion following injection of cultured chondrocytes and suturing of periosteal graft in place.

REFERENCES

1. Cahill B. Osteochondritis dissecans of the knee: treatment of juvenile and adult forms. *J Am Acad Orthop Surg.* 1995;3(4):237–247. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/10795030>
2. Dettlerline A, Goldstein J, Rue J, et al. Evaluation and treatment of osteochondritis dissecans lesions of the knee. *J Knee Surg.* 2008;21(2):106–115.
3. Ananthaharan A, Randsborg PH. Epidemiology and patient-reported outcome after juvenile osteochondritis dissecans in the knee. *Knee.* 2018;25(4):595–601. doi:10.1016/j.knee.2018.02.005.
4. Hughston J, Hergenroeder P, Courtenay B. Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am.* 1984;66(9):1340–1348.
5. Garrett J. Fresh osteochondral allografts for treatment of articular defects in osteochondritis dissecans of the lateral femoral condyle in adults. *Clin Orthop Relat Res.* 1994;303:33–37.
6. Bradley J, Dandy D. Osteochondritis dissecans and other lesions of the femoral condyles. *J Bone Joint Surg Br.* 1989;71(3):518–522.
7. Kocher M, Micheli L, Yaniv M, et al. Functional and radiographic outcome of juvenile osteochondritis dissecans of the knee treated with transarticular arthroscopic drilling. *Am J Sports Med.* 2001;29(5):562–566.
8. Bauer M, Jonsson K, Lindén B. Osteochondritis dissecans of the ankle. A 20-year follow-up study. *J Bone Joint Surg Br.* 1987;69(1):93–96.
9. Cahill B. Editorial commentary: current concepts review. Osteochondritis dissecans. *J Bone Joint Surg Am.* 1997;79(3):471–472.
10. Fowler J, Wicks M. Osteochondritis dissecans of the lunate. *J Hand Surg [Am].* 1990;15(4):571–572.
11. Mitsunaga M, Adishian D, Bianco A Jr. Osteochondritis dissecans of the capitellum. *J Trauma.* 1982;22(1):53–55.
12. Pappas A. Osteochondrosis dissecans. *Clin Orthop Relat Res.* 1981;158:59–69.
13. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. *J Bone Joint Surg Am.* 1977;59(6):769–776.
14. Mandelbaum B, Browne J, Fu F, et al. Articular cartilage lesions of the knee. *Am J Sports Med.* 1998;26(6):853–861.
15. Flynn J, Kocher M, Ganley T. Osteochondritis dissecans of the knee. *J Pediatr Orthop.* 2004;24(4):434–443.
16. Pascual-Garrido C, Moran CJ, Green DW, et al. Osteochondritis dissecans of the knee in children and adolescents. *Curr Opin Pediatr.* 2013;25(1):46–51. doi:10.1097/MOP.0b013e32835adbf5.
17. Ewing J, Voto S. Arthroscopic surgical management of osteochondritis dissecans of the knee. *Arthroscopy.* 1988;4(1):37–40.
18. Morelli M, Poitras P, Grimes V, et al. Comparison of the stability of various internal fixators used in the treatment of osteochondritis dissecans—a mechanical model. *J Orthop Res.* 2007;25(4):495–500.
19. Wouters D, Bos R, Mouton L, et al. The meniscus Arrow or metal screw for treatment of osteochondritis dissecans? In vitro comparison of their effectiveness. *Knee Surg Sports Traumatol Arthrosc.* 2004;12(1):52–57.
20. Simonian P, Sussmann P, Wickiewicz T, et al. Contact pressures at osteochondral donor sites in the knee. *Am J Sports Med.* 1998;26(4):491–494.
21. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. *J Bone Joint Surg Am.* 2003;85-A(suppl 2):25–32.
22. Gross A. Repair of cartilage defects in the knee. *J Knee Surg.* 2002;15(3):167–169.
23. Hefti F, Beguiristain J, Krauspe R, et al. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B.* 1999;8(4):231–245.
24. Scioscia T, Giffin J, Allen C, et al. Potential complication of bioabsorbable screw fixation for osteochondritis dissecans of the knee. *Arthroscopy.* 2001;17(2):E7.
25. Crawford E, Emery R, Aichroth P. Stable osteochondritis dissecans—does the lesion unite? *J Bone Joint Surg Br.* 1990;72(2):320.
26. Twyman R, Desai K, Aichroth P. Osteochondritis dissecans of the knee. A long-term study. *J Bone Joint Surg Br.* 1991;73(3):461–464.
27. Guhl J. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res.* 1982;167:65–74.
28. Mandelbaum BR. Editorial commentary: focal cartilage defects in young patients indicate autologous chondrocyte implantation sooner rather than later. *Arthroscopy.* 2016;32(9):1917–1918. doi:10.1016/j.arthro.2016.07.011.
29. Anderson A, Pagnani M. Osteochondritis Dissecans of the femoral condyles: long-term results of excision of the fragment. *Am J Sports Med.* 1997;25(6):830–834.
30. Cain E, Clancy W. Treatment algorithm for osteochondral injuries of the knee. *Clin Sports Med.* 2001;20(2):321–342. doi:10.1016/S0278-5919(05)70309-4.
31. Kocher M, Tucker R, Ganley T, et al. Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med.* 2006;34(7):1181–1191.
32. Louisia S, Beaufils P, Katabi M, et al. Transchondral drilling for osteochondritis dissecans of the medial condyle of the knee. *Knee Surg Sports Traumatol Arthrosc.* 2003;11(1):33–39.
33. Berna-Serna J, Martinez F, Reus M, et al. Osteochondritis dissecans of the knee: sonographically guided percutaneous drilling. *J Ultrasound Med.* 2008;27(2):255–259.
34. Kouzelis A, Plessas S, Papadopoulos A, et al. Herbert screw fixation and reverse guided drillings, for treatment of types III and IV osteochondritis dissecans. *Knee Surg Sports Traumatol Arthrosc.* 2006;14(1):70–75.
35. Lebolt J, Wall E. Retroarticular drilling and bone grafting of juvenile osteochondritis dissecans of the knee. *Arthroscopy.* 2007;23(7):791–794.
36. Gomoll A, Flik K, Hayden J, et al. Internal fixation of unstable Cahill type-2C osteochondritis dissecans lesions of the knee in adolescent patients. *Orthopedics.* 2007;30(6):487–490.
37. Miniaci A, Tytherleigh-Strong G. Fixation of unstable osteochondritis dissecans lesions of the knee using arthroscopic autogenous osteochondral grafting (mosaicplasty). *Arthroscopy.* 2007;23(8):845–851.
38. Lewis P, McCarty LI, Kang R, et al. Basic science and treatment options for articular cartilage injuries. *J Orthop Sports Phys Ther.* 2006;36(10):717–727.
39. McCarty L. Primary repair of osteochondritis dissecans in the knee. In: Cole B, Sekiya J, eds. *Surgical Techniques of the Shoulder, Elbow and Knee in Sports Medicine.* Philadelphia, PA: Saunders Elsevier; 2008.
40. Steadman JR, Briggs KK, Rodrigo JJ, et al. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. *Arthroscopy.* 2003;19(5):477–484. doi:10.1053/jars.2003.50112.

Q5

41. Frisbie DD, Lu Y, Kawcak CE, et al. In vivo evaluation of autologous cartilage fragment-loaded scaffolds implanted into equine articular defects and compared with autologous chondrocyte implantation. *Am J Sports Med.* 2009; 37(1_suppl):71S–80S. doi:10.1177/0363546509348478.
42. Minas T, Peterson L. Advanced techniques in autologous chondrocyte transplantation. *Clin Sports Med.* 1999;18(1):13–44.
43. Cotter EJ, Frank RM, Wang KC, et al. Clinical outcomes of osteochondral allograft transplantation for secondary treatment of osteochondritis dissecans of the knee in skeletally mature patients. *Arthroscopy.* 2018;34(4):1105–1112. doi:10.1016/j.arthro.2017.10.043.
44. Kramer DE, Yen YM, Simoni MK, et al. Surgical management of osteochondritis dissecans lesions of the patella and trochlea in the pediatric and adolescent population. *Am J Sports Med.* 2015;43(3):654–662. doi:10.1177/0363546514562174.
45. Friederichs M, Greis P, Burks R. Pitfalls associated with fixation of osteochondritis dissecans fragments using bioabsorbable screws. *Arthroscopy.* 2001;17(5):542–545.
46. Kivistö R, Pasanen L, Leppilahti J, et al. Arthroscopic repair of osteochondritis dissecans of the femoral condyles with metal staple fixation: a report of 28 cases. *Knee Surg Sports Traumatol Arthrosc.* 2002;10(5):305–309.
47. Denoncourt P, Patel D, Dimakopoulos P. Arthroscopy update #1. Treatment of osteochondritis dissecans of the knee by arthroscopic curettage, follow-up study. *Orthop Rev.* 1986;15(10):652–657.
48. Anderson A, Richards D, Pagnani M, et al. Antegrade drilling for osteochondritis dissecans of the knee. *Arthroscopy.* 1997;13(3):319–324.
49. Makino A, Muscolo D, Puigdevall M, et al. Arthroscopic fixation of osteochondritis dissecans of the knee: clinical, magnetic resonance imaging, and arthroscopic follow-up. *Am J Sports Med.* 2005;33(10):1499–1504.
50. Dines JS, Fealy S, Potter HG, et al. Outcomes of osteochondral lesions of the knee repaired with a bioabsorbable device. *Arthroscopy.* 2008;24(1):62–68. doi:10.1016/j.arthro.2007.07.025.
51. Nakagawa T, Kurosawa H, Ikeda H, et al. Internal fixation for osteochondritis dissecans of the knee. *Knee Surg Sports Traumatol Arthrosc.* 2005;13(4):317–322.
52. Weckström M, Parviainen M, Kiuru M, et al. Comparison of bioabsorbable pins and nails in the fixation of adult osteochondritis dissecans fragments of the knee: an outcome of 30 knees. *Am J Sports Med.* 2007;35(9):1467–1476.
53. Ishikawa M, Nakamae A, Nakasa T, et al. Limitation of in-situ arthroscopic fixation for stable juvenile osteochondritis dissecans in the knee. *J Pediatr Orthop B* 2018;27(6):516–521. doi:10.1097/BPB.0000000000000531.
54. Kubota M, Ishijima M, Ikeda H, et al. Mid and long term outcomes after fixation of osteochondritis dissecans. *J Orthop.* 2018;15(2):536–539. doi:10.1016/j.jor.2018.01.002.
55. Wu IT, Custers RJH, Desai VS, et al. Internal fixation of unstable osteochondritis dissecans: do open growth plates improve healing rate? *Am J Sports Med.* 2018;46(10):2394–2401. doi:10.1177/0363546518783737.
56. Chadli L, Steltzlen C, Beaufils P, et al. Neither significant osteoarthritic changes nor deteriorating subjective outcomes occur after hybrid fixation of osteochondritis dissecans in the young adult. *Knee Surg Sports Traumatol Arthrosc.* 2018;1–5. doi:10.1007/s00167-018-5025-0.
57. Gudas R, Stankevicius E, Monastyreckiene E, et al. Osteochondral autologous transplantation versus microfracture for the treatment of articular cartilage defects in the knee joint in athletes. *Knee Surg Sports Traumatol Arthrosc.* 2006;14(9):834–842.
58. McCulloch P, Kang R, Sobhy M, et al. Prospective evaluation of prolonged fresh osteochondral allograft transplantation of the femoral condyle: minimum 2-year follow-up. *Am J Sports Med.* 2007;35(3):411–420.
59. Knutsen G, Drogset J, Engebretsen L, et al. A randomized trial comparing autologous chondrocyte implantation with microfracture. *J Bone Joint Surg Am.* 2007;89(10):2105–2112.
60. Outerbridge H, Outerbridge A, Outerbridge R. The use of a lateral patellar autologous graft for the repair of a large osteochondral defect in the knee. *J Bone Joint Surg Am.* 1995;77(1):65–72.
61. Emmerson BC, Görtz S, Jamali AA, et al. Fresh osteochondral allografting in the treatment of osteochondritis dissecans of the femoral condyle. *Am J Sports Med.* 2007;35(6):907–914. doi:10.1177/0363546507299932.
62. Convery F, Meyers M, Akeson W. Fresh osteochondral allografting of the femoral condyle. *Clin Orthop Relat Res.* 1991;273:139–145.
63. Sadr KN, Pulido PA, McCauley JC, et al. Osteochondral allograft transplantation in patients with osteochondritis dissecans of the knee. *Am J Sports Med.* 2016;44(11):2870–2875. doi:10.1177/0363546516657526.
64. Peterson L, Minas T, Brittberg M, et al. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation: results at two to ten years. *J Bone Joint Surg Am.* 2003;85(suppl 2):17–24.
65. Cole BJ, Lee SJ. Complex knee reconstruction: articular cartilage treatment options. *Arthroscopy.* 2003;19(10 suppl 1):1–10. doi:10.1016/j.arthro.2003.09.025.

Queries

- [Q1] Please check whether the edits made to the sentence beginning "Neither the literature..." convey the intended meaning.
- [Q2] Please check whether permission is needed for the source line in Table 45-1.
- [Q3] Please check whether the edit made to the sentence beginning "Partial weight bearing..." is okay.
- [Q4] Please check for missing text after "either failed" in the sentence beginning "The study noted 24%...".
- [Q5] Please check the publisher name in reference 39.

Copyright © 2019 Wolters Kluwer, Inc. Unauthorized reproduction of the content is prohibited.