Osteochondritis Dissecans of the Knee

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Osteochondritis dissecans (OCD) is a relatively common cause of knee pain and dysfunction in children and adolescents. OCD is an acquired, potentially reversible idiopathic lesion of subchondral bone resulting in delamination and sequestration with or without articular cartilage involvement and instability.[11,15,26,31,37,52,65] Etiology of OCD remains speculative.[11,15,26,52] Knee OCD classifications are based on anatomic location, surgical appearance, scintigraphic findings, and chronological age.[11,12,15,26,29,52] OCD is subcategorized into juvenile and adult forms depending on distal femoral physis maturity. The majority of cases of adult OCD are thought to be due to persistence of an unresolved juvenile OCD lesion although de novo adult OCD lesions have been described.[11] Juvenile OCD has a better prognosis than adult OCD.[13,27,29,59,63] Adult OCD and juvenile OCD lesions which do not heal have potential for later sequelae.[47,62]

Juvenile OCD management is controversial. Nonoperative initial management is indicated for stable lesions in skeletally immature patients.[11,13,14,26] Operative treatment is indicated for detached or unstable lesions, a patient approaching epiphyseal closure, and failure of nonoperative management.[11]

We will review etiology, clinical presentation, diagnostic evaluation, nonoperative, and operative treatments and outcomes of knee OCD.

ETIOLOGY

OCD etiology remains enigmatic. Inflammation, genetics, ischemia, ossification, and repetitive trauma have been hypothesized causes. Terminology regarding osteochondral lesions of the knee overlaps, and this imprecision contributes to inconsistency regarding the diagnosis, management, and prognosis of OCD. OCD lesions may appear as acute osteochondral fractures, chondral injuries, and osteonecrosis.

An inflammatory etiology was suggested by König in 1887 who termed the condition "osteochondritis dissecans."[39] Further inquiries did not support inflammation as a cause of OCD. OCD was ascribed to an ossification abnormality of the distal femoral epiphysis by Ribbing in 1955 but no follow-up data were presented.[46] Abnormalities in ossification are not thought to be the etiology of OCD. Some incidentally found lateral femoral condyle lesions that resolve spontaneously in younger children may represent ossification variants. Ischemia was proposed as an etiology of OCD by Green and Banks based on anatomic and histologic findings.[27] Additional histopathologic inquiry failed to find avascular necrosis of the OCD fragment or a relative ischemic watershed of the lateral aspect of the medial femoral condyle.[14,55,56,57] Families with a genetic predisposition to OCD have been described. Mubarak and Carroll reported 12 family members over four generations who had OCD.[47] Petrie, in contrast, found OCD in only one of 86 first-degree relatives. Current thought holds that the common form of OCD is not familial.[51]

Another proposed etiology of OCD is repetitive, submaximal trauma. Fairbanks proposed that "violent rotation inwards of the tibia, driving the tibial spine against the inner condyle" caused OCD.[41] Anterior tibial spine impingement is not considered to be the etiology of lesions in the most common location (postero-lateral aspect of the medial femoral condyle), but the frequent occurrence of OCD in patients involved in sports with repetitive impact suggests a repetitive trauma etiology of unknown mechanism. Repetitive trauma perhaps induces a stress reaction which may progress to a stress fracture of underlying subchondral bone. If repetitive loading persists and exceeds the ability of the subchondral bone to heal, necrosis of the fragment may occur and lead to fragment dissection, separation, and non-union.

Knee OCD lesions are most commonly (70%–80%) seen in the "classic" or extended classic location of the posterolateral aspect of the medial femoral condyle. Infer-central lateral condylar lesions are seen in 15%–20% of cases. Patellar involvement is uncommon (5%–10%) and located in the inferior pole in most of cases. Femoral trochlear lesions account for <1% of cases.

Exact prevalence of OCD is unknown. Hughston reported a prevalence of 15–21 cases per 100,000.[30] Linden re-

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ported a prevalence of 18 per 100,000 in females and 29 per 100,000 in males.[1] Prevalence of OCD appears to be increasing secondary to more participation at higher competitive levels of sports by children at younger ages.[11] Mean age of OCD onset seems to be decreasing and increased number of girls now have involvement.[1] Increased acknowledgment of serious knee pathology in the pediatric population and widespread use of MRI and arthroscopy over the past decade have resulted in greater recognition of OCD lesions. Contemporaneous overtraining consequences of youth sports such as loss of free play, early sport specialization, multiple leagues in a single sport, and intensive training may also be contributing factors.

CLINICAL PRESENTATION

Most children and adolescents with osteochondritis dissecans have a stable lesion and presenting complaints are generally non-specific. The most common complaint is aching and activity-related knee pain localized to the anterior knee. Symptoms are similar to complaints heard for other causes of anterior knee pain. There may be pain when climbing hills or stairs. There is usually not a sense of knee instability or mechanical complaints.

On physical examination, children and adolescents with stable osteochondritis dissecans lesions may walk with an antalgic gait. A point of maximum tenderness can often be palpated over the anterior medial aspect of the knee with varying amounts of knee flexion. This area will correspond to the lesion, most commonly on the lateral aspect of the distal medial femoral condyle. In stable lesions, there is usually not an effusion, crepitus, or pain through a range of normal motion. Loss of quadriceps definition may be noted in long-standing cases.

Mechanical symptoms are more pronounced with unstable lesions. An antalgic gait is common, and there is usually a knee effusion, possibly associated with crepitus with motion. Both knees should be examined since the condition is bilateral in 20%–25% of cases. If bilateral, involvement magnitude is not symmetrical.

DIAGNOSTIC STUDIES

Successful imaging characterizes the lesion, determines prognosis of nonoperative management, and predicts ultimate status of the lesion. Because success of nonoperative management is unpredictable in juvenile osteochondritis dissecans, many studies have investigated various OCD imaging protocols. An ideal imaging strategy would guide the surgeon in determining which cases should be treated surgically and which cases would heal with nonoperative treatment. Technetium bone scanning, and, more recently, MRI and MR arthrography have been studied, but to date, no single imaging protocol reliably predicts success of nonoperative management.

Initial plain radiographs should include anteroposterior and lateral views of the knee (Fig. 1). Tunnel views are also valuable as an OCD in the classic location may be difficult to see on an anteroposterior view (Fig. 2). Skyline view should be added when patellar OCD is a possibility. The purpose of plain radiographs is to characterize and localize the lesion, rule out other bony pathology, and evaluate skeletal maturity. In children less than 7 years old, irregularities of the distal femoral epiphyseal ossification center may simulate OCD, but these asymptomatic sites are anatomic variants of normal ossification. Lesion location can be described as per Cahill and Berg[12] (Fig. 1) and an estimate of size obtained from the radiographs.

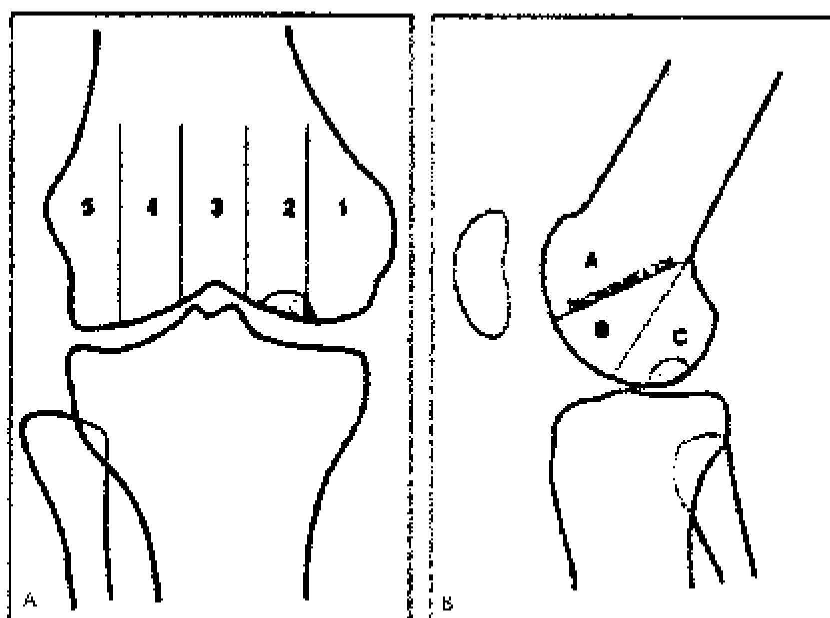

FIGURE 1. Anatomic classification of knee OCD based on lesion location on AP (A) and lateral (B) radiographs.

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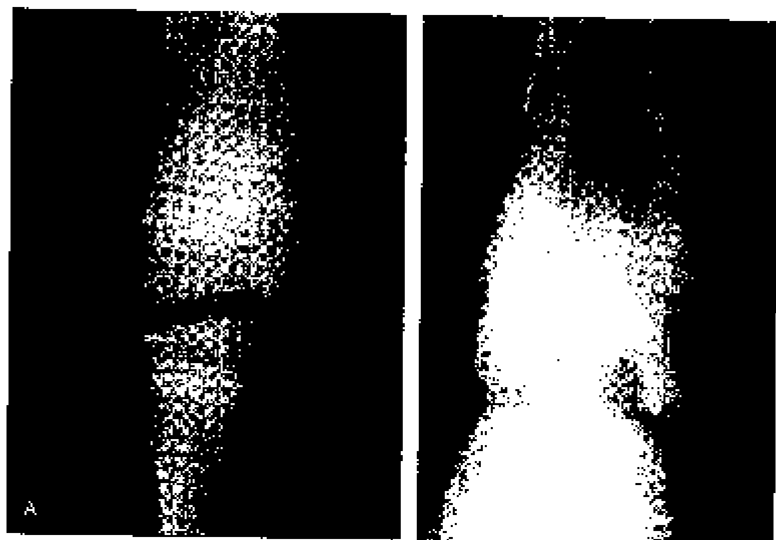

FIGURE 2. AP (A) and notch (B) radiographs of a juvenile OCD lesion of the medial femoral condyle. The lesion can be difficult to see on an AP radiograph and is often more apparent on the notch view which images the posterior aspect of the femoral condyle with knee flexion.

MR imaging has become a routine part of the diagnostic evaluation of osteochondritis dissecans.[20] MRI can give an accurate estimation of the size of the lesion and status of cartilage and subchondral bone (Figs. 3 and 4). Extent of bony edema, appearance of a high signal zone beneath the fragment, and presence of loose bodies are additional important MRI findings (Table 1).

For more than a decade, MR imaging results have been studied extensively with hope that certain findings would have definitive prognostic value for healing with nonoperative treatment.[9] De Smet et al[19] described 4 MRI criteria on T2 weighted images: (1) a line of high signal intensity at least 5 mm in length between the OCD lesion and underlying bone, (2) an area of increased homogeneous signal at least 5 mm in diameter beneath the lesion, (3) a focal defect of 5 mm or more in the articular surface, and (4) a high signal line traversing the subchondral plate into the lesion. Of these signs, De Smet et al found that a high signal line behind the fragment was most predictive and found in 72% of unstable lesions. Pill et al[35] attempted to predict success of nonoperative treatment using MRI and clinical criteria. They applied De Smet's 4 signs and found the high signal line the most common sign in patients who failed nonoperative treatment. Lesion size and patient maturity were also important predictors of failure of nonoperative treatment. O'Connor et al[29] compared MRI and arthroscopic findings, focusing specifically on prognostic value of De Smet's high signal line behind the fragment. These authors and others believe this high signal line represents either healing vascular granulation tissue or synovial fluid collected beneath the subchondral bone implying a break in the articular surface.

FIGURE 3. AP radiograph (A) and coronal (B) MRI of a stage 2 juvenile OCD lesion of the medial femoral condyle. The lesion is clearly demarcated from underlying subchondral bone without evidence of healing and an intact articular mantle.

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FIGURE 4. Lateral radiograph (A) and sagittal (B) MRI of a stage 3 juvenile OCD lesion of the medial femoral condyle. The lesion is clearly demarcated from underlying subchondral bone with apparent anterior separation of the articular surface.

In this study, investigators improved staging accuracy from 45% to 85% when they interpreted high signal line on T2 images a predictor of instability only when accompanied by a breach in articular cartilage seen on T1 images.

Several studies showed un-enhanced MRI does not have definitive prognostic value in juvenile osteochondritis dissecans. Other investigators explored the value of gadolinium MRI enhancement. After intravenous gadolinium administration, Bohndorf[9] found enhancement of high signal line behind the fragment indicated healing granulation tissue not synovial fluid. In contrast, Vonstein et al[64] showed no correlation between gadolinium enhancement and healing in juvenile osteochondritis dissecans cases. These investigators found that lesion size was the main determinant of healing. Although Kramer and colleagues[40] did not look at prognostic value in terms of healing, they determined that MR arthrography with gadolinium could reliably show loss of continuity of articular cartilage mantle.

Technetium bone scans have been used to assess healing potential of OCD lesions. Cahill and Berg[12] proposed static serial qualitative technicium bone scans every 6 weeks until evidence of healing occurred (Table 2). Unfortunately, the isotopic tracer remains in the area in question for a significant time, even after healing which makes interpretation difficult. Litchman et al[43] found in a small number of mature patients that those with more than 2 months of symptoms from an OCD who had increased blood flow on quantiative technicium scans healed their lesions spontaneously. Paletta et al[51] reviewed quantitative bone scans in a small series (12 patients) and found increased activity predicted healing in 100% of patients with open femoral physes but not in adolescents with closing physes. Unfortunately, it is this latter group in whom healing is most difficult to predict. Serial bone scanning has not been widely adopted in the management of OCD lesions, perhaps because of time required for the study, need for intravenous access, and perceived risk of the isotope as well as the emergence of MR imaging.

Considering published results to date, current diagnostic imaging recommendations for OCD are initial AP, lateral and tunnel, and skyline radiographs. An MRI is often obtained, to assess lesion size, status of cartilage and subchondral bone, presence of high signal zone beneath the lesion, extent of bony edema, as well as possible presence of loose bodies or other

TABLE 1. MRI Classification of JOCD Lesions[29]

Stage I: Small change of signal without clear margins of fragment.

Stage II: Osteochondral fragment with clear margins without fluid between fragment and underlying bone.

Stage III: Fluid is partially visible between fragment and underlying bone.

Stage IV: Fluid completely surrounds the fragment but the fragment is in situ.

Stage V: Fragment is completely detached and displaced (loose body).

TABLE 2. Bone Scan Classification of JOCD Lesions[11]

Stage 0: Normal radiographic and scintigraphic appearance.

Stage I: Lesion is visible on plain radiographs but bone scans reveal normal findings.

Stage II: Scan reveals increased uptake in the area of the lesion.

Stage III: In addition, there is increased isotopic uptake in the entire femoral condyle.

Stage IV: In addition, there is uptake in the tibial plateau opposite the lesion.

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knee pathology. Smaller lesions with intact cartilage are much more likely to respond to nonoperative treatment, especially in skeletally immature patients. Patients with unstable lesions, loose bodies, torn menisci, or other intra-articular pathology warrant initial arthroscopic evaluation and treatment.

NONOPERATIVE MANAGEMENT

Since the natural history of stable osteochondritis dissecans lesions is generally favorable in a child with open physes, there is widespread agreement that initial nonoperative management is indicated.[53] A debate exists about therapeutic or detrimental effects of immobilization. This controversy centers on which tissue is considered most important in healing. Advocates who focus on subchondral bone argue that initially the knee should be protected in a cast or knee immobilizer and treated as a fracture. Conversely, authors focused on articular cartilage cite the value of continuous passive motion for cartilage health. Because failure of cartilage surface is subsequent to failure of underlying bone, most have embraced some type of altered activity and/or immobilization protocol.

Immobilization can be successfully achieved in a cast, brace, or standard knee immobilizer. Some authors prefer partial weight bearing in a cylinder cast in slight flexion which is thought to allow some compressive forces across the lesion while minimizing shear. This protocol can also usually be accomplished with a standard knee immobilizer. Bathing and other activities are easier, but compliance with full-time immobilizer use by a young athlete can be a problem. Some authors recommend a hinged, unloader-type brace that allows motion. This treatment's efficacy has not been proven, and problems with compliance and expense exist. Further data is needed to determine optimal immobilization protocol.

Our recommended nonoperative management protocol is in three phases. The first phase involves knee immobilization for 6 weeks with crutch-protected, partial weight-bearing gait. The child should be pain free at the end of this period. Radiographs are repeated. In phase 2 (weeks 6–12), weight bearing as tolerated is permitted without immobilization. A rehabilitation program is initiated emphasizing knee range of motion and low-impact quadriceps and hamstring strengthening exercises. If the patient remains pain free, phase 3 begins at 3 months after diagnosis. This phase includes supervised initiation of running, jumping, and cutting sports readiness activities. High impact and shear activities are restricted until the child has had several months of pain free, low-impact conditioning and radiographs document healing. An MRI may be repeated in phase 3 to assess healing.

If symptoms return or radiographs show lesion progression, repeat immobilization is considered. Immobilization alone is often successful in juvenile osteochondritis dissecans but may be intolerable to a young athlete and their parents. Dealing with these impatient and frustrated young patients includes counseling on risks and benefits of continued nonoperative treatment versus moving on to surgical management.

Operative Management

Despite patients with juvenile osteochondritis dissecans having a good prognosis for healing, not all lesions heal in skeletally immature knees. Operative treatment should be considered in patients with detached or unstable lesions and in those patients approaching physeal closure whose lesions have been unresponsive to nonoperative management.[11,20,28,31]

OCD affects subchondral bone and can secondarily compromise articular cartilage making the goal of treatment healing the junctional subchondral bone sequestrum. Goals of operative treatment also include maintenance of joint congruity, rigid fixation of unstable fragments, and repair of osteochondral defects.[59] To achieve the latter, an ample supply of surrounding stable articular cartilage and a stable construct of subchondral bone, calcified tidemark, and repair cartilage with viability and biomechanical properties closely resembling native hyaline cartilage are needed.

For cases that fail nonoperative treatment but have a stable lesion and intact articular surface, arthroscopic drilling of the lesion creates channels for potential revascularization and healing (Fig. 5). Drilling options include passing through the epiphysis without articular penetration or via transarticular drilling. Drilling through the epiphysis avoids articular surface violation but is associated with technical challenges of maintaining drill depth and placement accuracy. Transarticular drilling is technically straightforward, although this method creates articular cartilage channels which heal with fibrocartilage.[6] Posterior lesions may be difficult to access with this method and are easily approached by the transepiphyseal technique.

A number of authors found arthroscopic transarticular drilling effective in treatment of OCD lesions in skeletally im-

FIGURE 5. Arthroscopic image of retrograde transarticular drilling of a stable OCD lesion of the medial femoral condyle with an intact articular surface.

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FIGURE 6. Lesions of the lateral femoral condyle (A) and trochlea (B) are less likely to heal with nonoperative treatment or arthroscopic drilling than medial femoral condyle lesions.

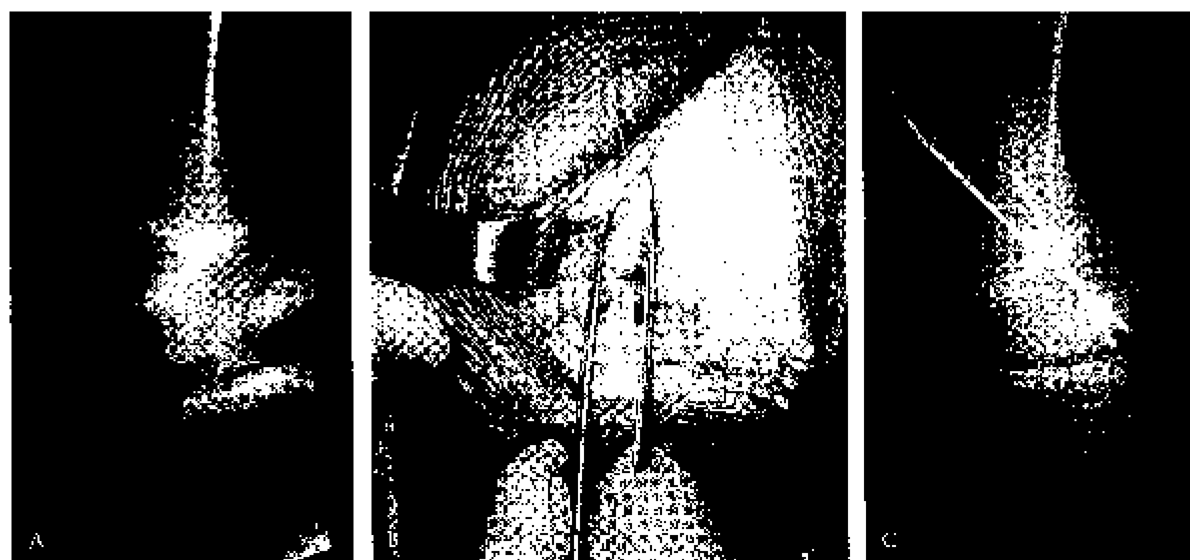

FIGURE 7. Unstable OCD lesion in a patient approaching skeletal maturity (A) treated with arthrotomy (B), bone grafting, and K wire fixation (C) 3 months postoperative radiograph.

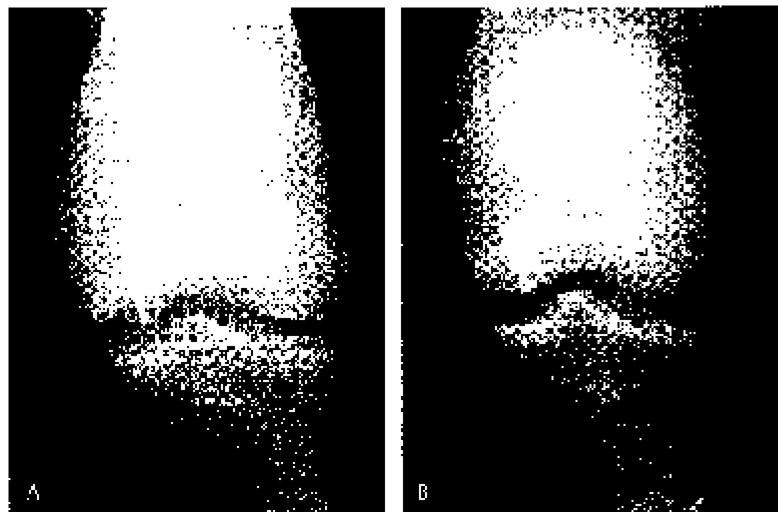

FIGURE 8. Unstable OCD lesion in a patient approaching skeletal maturity treated with arthroscopic fixation using a variable pitch screw (A) 3 weeks postoperative; (B) 3 months postoperative radiographs.

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mature patients. Aglietti et al[1] noted radiographic healing after drilling in 16 knees in 14 patients. All patients were asymptomatic at an average follow-up of 4 years.[1] Bradley and Dandy[10] performed this technique in patients with OCD lesions in the classic site and noted radiographic healing and pain relief in 9 of 11 knees within 1 year. One knee was healed within 2 years, and a nonunion with loose body formation developed in one knee.[10] Anderson and colleagues[6] performed transarticular drilling in 17 patients (20 knees) with open physes and in 4 patients with closed physes. In the skeletally immature group, 18 of 20 lesions healed. In the small skeletally mature group, 2 of 4 healed at an average follow up of 5 years.[6] Transarticular drilling was performed on 51 knees in 49 patients up to 18 years of age at the Children's Hospital of Philadelphia. Drilling was effective in skeletally immature patients and was curative in 83% of adolescents with open physes in contrast to 75% of adolescents with closed physes. Factors associated with inadequate healing despite drilling included lesions in non-classic locations (Fig. 6), multiple lesions and patients with underlying medical conditions.[23] Kocher et al reviewed functional and radiographic outcomes using this technique[38] in 23 skeletally immature patients (30 knees) with lesions at the classic location at an average follow-up of 3.9 years. There was significant improvement in the mean Lysholm score, and radiographic healing was achieved in all patients at an average of 4.4 months after drilling. Younger age was noted to be an independent multivariate predictor of Lysholm score using linear regression analysis.

In cases with flap lesions, fibrous tissue found between fragments should be removed. Debridement of significant portions of bone from the fragment and subchondral base of the lesion should be avoided. If partially unstable lesions have subchondral bone loss, autogenous bone graft is packed into the crater prior to fragment reduction and fixation (Fig. 7). In patients with unstable lesions with subchondral bone attached with an anatomic match between defect and fragment, fixation can be performed by a variety of arthroscopic or open methods. Rapid relief of pain postoperatively has led some authors to theorize that increased pressure at the line of separation between the fragment and the epiphysis may be a source of pain.[10,37,61] Navarro et al recently reported use of tibial metaphyseal bone strips to treat partially displaced OCD lesions.[48] All 11 patients in their series returned to strenuous activities. Herbert screws and cannulated screws have been used successfully (Figs. 8 and 9), with second surgeries required for removal.[17,68] Kivisto et al reported on use of arthroscopically placed metal staples which were thought to not require removal but broken staples were seen in 9 of 25 knees.[51]

FIGURE 9. Loose body lesion treated with arthrotomy, fragment reshaping, bone grafting, and fixation.

Despite use of a variety of bio-absorbable screws and pins (Fig. 10) and bone and osteochondral plugs, a number of authors have described complications associated with these treatments.[18,22,33,58] Friederichs and Scioscia, in separate reports, noted loosing and failure of bio-absorbable screws that had backed out causing damage to adjacent articular surfaces and unabsorbed screw heads found as intraarticular loose bodies.[22,58] Kim and Shin described donor site generated loose bodies as a complication of osteochondral allograft treatment of osteochondritis dissecans.[33]

A variety of techniques have been developed for treatment of large unsalvageable fragments (Fig. 11) in an attempt to replace the defect with a composite of subchondral bone calcified tidemark and overlying cartilage. Drilling, abrasion arthroplasty, and microfracture methods attempt to recruit plu-

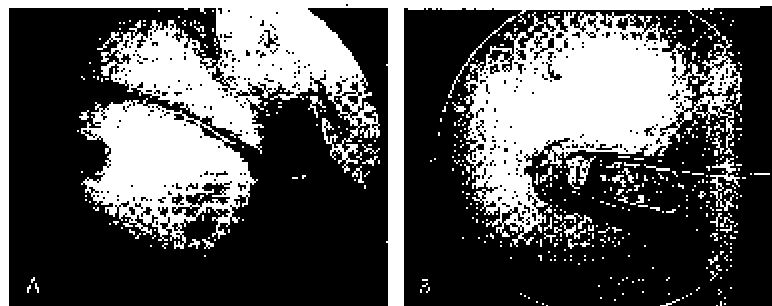

FIGURE 10. Bioabsorbable fixation for OCD lesions: (A) bioabsorbable pins and (B) bioabsorbable tack.

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FIGURE 11. Excision of a chronic loose body.

tipotential cells from marrow elements.[60] The recruited cells differentiate primarily into fibrocartilage that typically responds poorly to shear forces. Perhaps, small lesions in non–weight-bearing area can be resurfaced using these techniques. Results using these techniques for large lesions show deterioration with time due to decreased fibrocartilage resilience and stiffness.[45] Agletti et al felt that fragment removal and crater debridement alone was a viable option,[2,5] but, in adults, one grade worsening of Fairbanks radiographic changes was found in 45% of patients at an average follow-up of 9 years. Results of this technique were better in lesions less than 2 cm.

Transplanted periosteum with the cambium layer facing the defect has been used to produce a cartilaginous extracellular matrix. Neidermann reported successful results after periosteal transplantation for knee OCD at a very brief 1 year follow-up. Angermann et al had disappointing results at 6- to 9-year follow-up using this method in 14 patients with adult OCD.[55] Madsen et al studied long-term results of periosteal transplantation without chondrocyte grafting in knee OCD.[11] Median patient age was 19 years among the 18 patients studied. Eight patients required reoperations up to 8 years postoperatively.

Other techniques have been developed to address the inadequate structural properties of reparative fibrocartilage. Transplantation of autologous osteochondral plugs from non–weight-bearing regions of the knee been used for defect replacement in skeletally mature patients[7,8] Outerbridge reported good short-term results using osteochondral grafts harvested from the lateral facet of the patella in 10 adult patients with large femoral OCD lesions.[50] Yoshizumi et al reported on successful osteochondral graft treatment of osteochondritis dissecans lesions in 3 skeletally mature patients 18 years of age and younger[67] and noted potential disadvantages of donor site morbidity and incongruent articular fit and the advantages of biologic internal fixation.

Autologous chondrocyte implantation has been used to treat large isolated femoral defects in skeletally mature patients without lower extremity malalignment. Peterson et al reported results of this treatment of OCD at 2–10 years follow-up.[53] Mean patient age was 26.4 years in the 48 patients treated. Thirty-five patients had onset of osteochondritis dissecans as juveniles. These authors found an integrated nonarticular cartilage repair tissue had been formed and noted successful clinical results in over 90% of patients. Bentley et al performed a prospective randomized comparison trial and found autologous chondrocyte implantation had significantly superior outcomes over autologous plugs for osteochondral defects in adult knees.[4] Autologous chondrocyte implantation results were comparable with those in other studies. Mosaicplasty plug results deteriorated with time. King et al[34] evaluated autologous chondrocyte transplantation for treatment of large defects in articular cartilage of the distal femur in mature adolescents and noted short-term outcomes slightly better than previous reported results in adult patients.

Secondary reconstruction with bone-articular surface allografts has been successful in patients with major surface OCD defects. No long-term results in skeletally immature patients are available.[24] Logistical problems of storage and congruous placement are inherent to this method.

To date, results of all methods of articular resurfacing in skeletally immature patients with OCD must be considered preliminary and investigative. A treatment algorithm overview is provided in Figure 12.

SUMMARY

Knee OCD is seen with increased frequency in pediatric patients. Early recognition is essential. Stable juvenile OCD lesions with an intact articular surface have a high potential to heal with nonoperative treatment, the mainstay of which is cessation of repetitive impact loading. Value of adjunctive immobilization, protected weight bearing, and unloader bracing has not been established. Patients with stable lesions that have not healed with 6–9 months of nonoperative treatment should have consideration given to arthroscopic drilling to promote healing before the lesion progresses and requires more involved treatment with a less sanguine prognosis. MR imaging may allow early prediction of lesion healing potential. Unstable lesions

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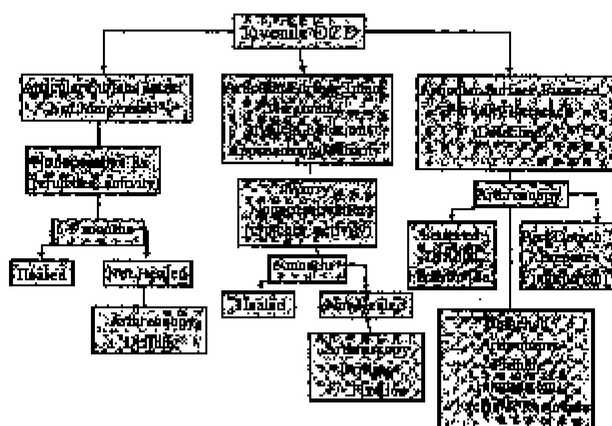

FIGURE 12. Algorithm for treatment of juvenile knee OCD.

and secondary loose bodies require fixation and possible bone grafting. Many unstable lesions will heal following stabilization, but long-term prognosis is not clear. Long established loosed fragments can be difficult to fix and have poor healing potential. Results of excision of large lesions from weight bearing zones, especially laterally, are poor. Chondral resurfacing techniques have limited long term data for cases of OCD in skeletally immature patients.

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