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SYMPOSIUM: OSTEOCHONDRITIS DISSECANS

A Review of Knowledge in Osteochondritis Dissecans: 123 Years of Minimal Evolution from König to the ROCK Study Group

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Abstract

Background Osteochondritis dissecans (OCD) was first described to provide an explanation for the nontraumatic development of loose bodies within a joint. Despite many reports on the subject, there remains no clear understanding of the etiology, natural history, or treatment.

Questions/purposes This review was undertaken to delineate (1) the etiology of OCD; (2) the presentation and locations; (3) the most appropriate imaging modalities; and (4) the most effective treatment strategies.

Methods We reviewed the English literature using a database compiled from a Medline search for "osteochondritis dissecans". We identified 1716 publications, 1246 of which were in English. After exclusions, we reviewed 748 articles and of these cited 85. The observations of each study were then synthesized into this report.

Results There appears to be no consensus concerning the etiology of OCD lesions. The presentations and locations are variable, but the knee, ankle, and elbow are most commonly involved. Although plain film assessment is important in OCD, there appears to be a trend toward the use of MRI, but the preferred sequences are in evolution. We found no consensus on the treatment of these lesions, related in part to the lack of agreement of methods for assessing outcomes.

Conclusions Despite more than a century of study, we have made little advancement in our understanding of OCD. A study group has been formed to address this issue and actively seeks to answer these unknown issues regarding OCD.

Introduction

Loose bodies within a joint were first described in 1870 by Paget [64]. In 1888, König (in a paper translated to English in this symposium) suggested three methods by which loose bodies could be created: (1) direct trauma with acute osteochondral fracture; (2) minimal trauma that develops into osteonecrosis and subsequent fragmentation; or (3) no evidence of trauma with spontaneous development that he coined osteochondritis dissecans (OCD) [21, 41].

In the past, many surgeons suggested skeletally immature patients (juvenile OCD) had a better prognosis, inconsistently defined as either radiographic healing or resolution of pain [5, 9, 14, 40, 47, 58]. However, despite differences in prognosis based on age, many of these authors believed juvenile and adult-onset OCD reflected the same pathologic processes but merely discovered at different points of skeletal maturity.

Although the exact pathophysiology is unknown, OCD is currently recognized as an acquired lesion of subchondral bone characterized by degrees of osseous resorption, collapse, and sequestrum formation with possible involvement of the articular cartilage through delamination

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unrelated to an acute osteochondral fracture of normal cartilage [14, 40]. This understanding of the end point of the disease process has led to many etiologies of OCD being postulated (particularly concerning the knee), including trauma [1, 3], inflammation [41, 70], genetics [58], vascular abnormalities [47, 48], and constitutional factors [9]; but, again, the cause remains unknown.

We examine the evolution of knowledge from the time of Professor König's first description until the present time when a study group (Research on OsteoChondritis of the Knee [ROCK]) was formed to examine over a century's worth of hypotheses, prospectively research the nature of OCD, and develop defined algorithms for treatment. The focus is on OCDs discovered during childhood and those involving the knee, although OCDs have been found in many other joints, including the elbow and ankle. We specifically assessed the literature to examine (1) the etiology of OCD; (2) the presentation and locations; (3) the most appropriate imaging; and (4) the most effective treatment strategies.

Search Strategy and Criteria

An initial PubMed and Medline search was performed of all manuscripts before the Fall of 2011 using the following key phrase: osteochondritis dissecans. This yielded 1716 publications, but after excluding non-English articles, this yielded 1246 manuscripts. We then excluded papers related to osteochondrosis, case reports, letters to the editor, and other nonscientific publications less review papers. This yielded 748 articles, all of which we reviewed the abstracts for content. Three hundred ten were excluded because they were animal studies unrelated to our key questions or they contributed limited data compared with similar papers that had a better methodology or sample size, unless they were of historical interest. Likewise, two general review papers were retained if they contributed some new data or hypothesis concerning our key questions, yielding 438 articles we reviewed in detail.

The 438 articles were reviewed with our four questions in mind. Each paper was earmarked concerning the presence, or absence, of an attempted answer to one of our four study questions and segregated into categories based on those earmarks. If the authors attempted to answer more than one of our study questions, then their paper was placed into as many categories as appropriate. For those articles answering the question of etiology, their hypotheses were recorded and their methodologies (retrospective review, prospective evaluation, or case series, etc) noted. Those papers discussing presentation and location were reviewed for those specific answers and the number of patients and methodologies recorded. Articles discussing radiographic assessment were divided among plain film, MRI, and other. Each article was then assessed based on sample size and results recorded for comparison. Finally, the articles dealing with treatment of OCD were categorized into nonoperative and operative and the various techniques were recorded along with outcomes. Methodology was also recorded to help determine inclusion in our study.

Once all the papers were reviewed in this fashion, we elected to discuss only the 85 papers that used the best methodology (if multiple papers discussed similar findings) or those papers with a unique perspective (despite possibly having poor methodology).

Etiology

Although no conclusive evidence has been discovered over the past 123 years of evaluation, many hypotheses have been presented and tested primarily by ex vivo histology (but discussed in detail subsequently). The potential etiologies include: inflammation, spontaneous osteonecrosis and vascular deficiency, genetics, and repetitive trauma.

König's originally postulated OCD was the result of inflammation, hence the coining of the term osteochondritis [41]. However, subsequent histologic studies have not supported an inflammatory etiology [2, 3]. Instead, these works as well as those cited subsequently demonstrate findings of necrosis within the OCD lesions rather than inflammation.

In 1953, Green and Banks [9, 28] proposed ischemia as the cause based on histologic samples harvested after surgical excision of loose bodies. Subsequently, Milgram [54] demonstrated revascularization in 50 lesions that were only partially attached. However, Yonetani et al. [84] found no osseous necrosis on microscopic examination in biopsy samples obtained from intact OCD lesions. Furthermore, Uozumi et al. [78] demonstrated an absence of subchondral bone in six of 11 biopsy samples and of the remaining five specimens with an osseous component, only two demonstrated no viable osteocytes. Therefore, it seems the necrosis found in OCD may be secondary to the actual detachment of the lesion rather than the underlying etiology of formation.

Many authors have suggested a familial inheritance, even to the point of describing it as a mild form of skeletal dysplasia with associated short stature [43, 58, 59, 68, 82]. An autosomal-dominant inheritance pattern is espoused by at least two separate authors [58, 68]. However, disputing the evidence of familial inheritance, Petrie [67] reported on a radiographic examination of first-degree relatives and discovered only 1.2% with an OCD. Although genetics may still play a role, the association remains elusive.

Repetitive trauma is currently the most commonly accepted etiology, but the nature of how and why is

unclear. In 1933, Fairbanks [19] proposed traumatic contact between the lateral aspect of the medial femoral condyle and the tibial spine as the cause of OCD. However, Fairbank's theory would only explain OCD lesions in the lateral aspect of the medial femoral condyle. The idea of repetitive trauma still holds our attention because subsequent studies have demonstrated an association between participation in sports and OCD, suggesting repetitive trauma with frequent involvement in athletic activity may be the cause. Aichroth [1] demonstrated 60% of the patients with OCD in his study were involved in high-level, competitive sports. Moreover, Linden [47] showed an association between the incidence of OCD and the increased involvement in organized sports in Sweden between 1965 and 1974. More recently, a multicenter study conducted by the European Pediatric Orthopedic Society demonstrated nearly 55% of the patients with OCD were either regularly active in sports or performed "strenuous athletic activity" [31]. Evidence of repetitive trauma as the etiology of OCD is lacking.

Several studies have noted associations between lateral femoral condyle OCD and discoid meniscus [56, 74, 85] and mechanical axis malalignment and the presence of OCD [33]. In fact, the positive association was present for varus malalignment and medial lesions as well as valgus malalignment for lateral lesions [33]. These findings suggest aberrant mechanical pressure on the condyles may be an etiology to the formation of OCD (at least in the knee).

One hypothesis concerning the etiology may unite all the previous evidence and it relates to the epiphyseal endochondral ossification. In 1937, Ribbing [69] presented a dialogue in a 107-page supplement to the journal Acta Radiologica during which he discussed abnormalities of endochondral ossification of the epiphysis. This discussion led Barrie [2, 3] to further define possible etiologies of OCD formation through an aberrant development of only a portion of the epiphyseal growth plate. The concept is that an OCD is slowly evolving with age of the patient. At an unspecified index time, there is an insult (single or repetitive) to the endochondral epiphyseal growth plate (this is the moment in which the hypothesis of trauma may converge). With skeletal development, the uninjured region of endochondral epiphyseal ossification continues to ossify unhindered creating an ever enlarging OCD, whereas the injured region either completely stops ossification or temporarily stops ossification. Barrie did not present any evidence to support this specific mechanism, but current unpublished data from the ROCK study group suggest this mechanism may be visible on MRI. It is most evident on T2 fat saturation sequences wherein the signal for the secondary physis appears disrupted at the margins of the OCD lesion. The permanent cessation of ossification may result in a completely cartilaginous OCD without endochondral ossification, whereas the temporary cessation of ossification may merely stall the endochondral ossification and that allows for either future partial ossification or complete ossification with time.

However, despite the gain of 123 years of knowledge, the ultimate etiology of OCD is yet to be determined. Perhaps the future will find that development of OCD is multifactorial or that there are multiple etiologies that may result in an OCD lesion.

Presentation and Location

Children (ie, those who are skeletally immature) with OCD may have two different presentations. The OCD may be an incidental radiographic findings in association with another unrelated injury or it may be the primary finding with clinically nonspecific activity-related knee pain with or without a notable history of trauma [26, 39]. Irregular ossification of the condyle may explain some of the asymptomatic, incidentally discovered OCD lesions and needs to be considered when making a clinical assessment of the young patient. Cahill and Ahten [10] reported 80% of the patients in their series had usually mild pain for an average of 14 months and a subtle or mild limp after activity reported by the parents. The pain experienced with OCD may be related to idiopathic adolescent knee pain rather than associated with the OCD or there may be substantial symptoms associated with the OCD lesion such as the mechanical symptoms of locking and popping. Effusion is present in less than 20% at presentation [31]. Wilson [81] described a test on physical examination to diagnose OCD: the knee is flexed to 90°, the tibia is externally rotated, and the knee is gradually extended to 30° of flexion. A positive test is characterized by pain over the anteromedial aspect of the knee as the knee is extended to 30° with relief of pain with internal rotation of the tibia. Anatomically, this maneuver is believed to cause impingement of the tibial spine on the lateral aspect of the medial femoral condyle consistent with the etiology hypothesis of Fairbanks described previously. The Wilson test is of limited diagnostic value with a reported positive test in only 16% of knees with radiographically proven OCD lesions [31].

The most common site of OCD development in the knee is the lateral aspect of the medial femoral condyle (51% involvement according to Hefti et al.) [31]. In their multicenter series of 509 knees in 452 patients they found other sites were involved with less frequency: 19% central medial femoral condyle, 17% lateral femoral condyle, 7% medial side of the medial femoral condyle, and 7% patella. Irregular ossification centers of the distal femoral condyle tend to be found more posterior on the condyle (although

they can be anywhere on the condyle) and are associated with younger age.

Imaging

The diagnosis of OCD is dependent on imaging, especially considering many lesions may be asymptomatic until separation or dislodging occurs. Most lesions can be identified by plain radiographs as long as four views are obtained, including AP, lateral, tunnel, and merchant views, although there is no English-based literature confirming the sensitivity or specificity. This provides visual access to the common locations for OCD lesions. Furthermore, since Cahill et al. [11, 23] demonstrated 16 of 76 patients had bilateral knee involvement, we believe it clinically acceptable to obtain contralateral films to evaluate for bilateral involvement.

The question of defining healing is important, yet the solution has not been fully resolved. From an imaging standpoint, it can be difficult just to determine if the lesion is actually an OCD. In light of the irregular centers of ossification, some authors have even speculated the improved prognosis seen in juvenile OCD is actually related to an incorrect diagnosis of OCD [24]. MRI can be helpful in the differentiation of these two lesion types. Nawata et al. [62] demonstrated MRI characteristics consistent with anomalous ossification; the low signal intensity of the lesion bone was similar to the surrounding subchondral bone and the high signal area demarcating the lesion was equal to the surrounding articular cartilage. Irregular ossification (ie, nonpathologic irregularity of ossification seen predominantly in children under the age of 10 years old) without dense bony margins can be followed without intervention if subsequent radiographs confirm the diagnosis through continued resolution of the irregularity. Radiographic assessment should demonstrate continued resolution, but if the lesion exhibits any radiographic signs of worsening (enlargement or an increasingly dense border), or the lesion fails to resolve as the child grows, then a diagnosis of OCD may be warranted.

Once an OCD has been formally diagnosed based on these criteria, then the next difficulty is defining healing. The literature over the past century has used plain radiology primarily to define healing of the lesion; however, the definition of healing has varied from author to author. Edmonds et al. discussed the various definitions [17], but briefly some authors define resolution as filling in of the cartilaginous lesion to osseous density or some percentage of that process. Others define radiographic healing by resolution or resorption of the dense margin defining the lesion. There is no direct evidence to support the use of the aforementioned definitions, and Wall et al. defined healing as the permanent resolution of symptoms regardless of any radiographic changes [80]. No one as yet correlated radiographic findings with healing, because healing has not yet been defined.

Over the last 123 years, the type of radiographic assessment has evolved based on the advances in technology. After plain film radiology, some authors have recommended the next evaluation should be scintigraphy because it provided information regarding blood flow to the lesion [8, 11, 50, 65]. However, it fails to provide detailed information about the state of affairs concerning the articular cartilage. Therefore, with the advent and subsequent development in MRI, this modality has become the diagnostic test of choice as a result of additional information concerning cartilage health, but no direct comparison between plain radiography and MRI has been performed [31]. MRI provides detailed images that provide a noninvasive method to assess the size, location, and character of the OCD lesion, including an estimation of stability (evidence of linear high-intensity signals on T2 sequences between the lesion and parent bone) and state of the articular cartilage (fissuring, thickness, water content, etc). An unstable lesion is defined by either fractured cartilage or separation of the underlying subchondral bone [79]. The question of stability appears to dictate the treatment and prognosis and, in general, stable lesions are believed to have a better likelihood of relief of symptoms and resolution of radiographic findings [12]. This is important because despite the lack of understanding the prognosis of OCD, some authors assume stable OCD lesions may be considered for nonoperative management, whereas unstable lesions are better treated with surgical intervention [12, 31]. Over the last decade, our understanding of MRI has evolved and we are developing a better understanding as to what the MR images are showing us concerning the ability to predict prognosis.

De Smet and colleagues [15, 16] described four criteria (on T2-weighted MR images) that correlate with instability when compared with arthroscopy findings: (1) a high signal line beneath the lesion; (2) a focal defect in the articular cartilage; (3) a fracture of the articular cartilage; and (4) the presence of subchondral cysts. In the aforementioned studies, the presence of a high T2 signal line between the lesion and the remainder of the normal bone was the most predictive factor of instability. However, the work of De Smet and his colleagues did not differentiate young and old patients. When Yoshida et al. [85] evaluated skeletally immature patients with this high T2 signal line separating the lesion, they could not predict stability accurately because the majority healed. Another study [63] suggested that if the criteria for instability included both the high T2 signal separating the lesion and a breach of the articular cartilage on T1-weighted images, then the accuracy in

predicting instability shifted from 45% to 85%. Recently, Kijowski et al. [36] examined the sensitivity and specificity of the criteria of De Smet et al. [16] for predicting arthroscopic instability (frank evidence of cartilage disruption or motion of the overlaying cartilage at the OCD) and applied them to adult OCD and to juvenile OCD. They reported that when all four criteria were applied together, there was 100% sensitivity and 100% specificity for instability in adult OCD and 100% sensitivity but only 11% specificity for instability in childhood OCD lesions. Thus, MRI should be considered a valuable tool for diagnosis, but it remains unclear whether MRI can accurately determine instability and prognosis.

Treatment

Two systematic reviews [12, 31] suggest most authors believe treatment should be based on skeletal maturity and lesion stability. A number of classification systems for OCD have included these two essential points [9, 15, 16, 30].

Some authors [9, 80] believe nonoperative management should be the first-line treatment for stable OCD lesions in children. The only consensus in regard to this modality is that if this treatment is elected, then the duration should be 3 to 6 months before opting for operative treatment [12, 39, 79, 80]. The actual definition of nonoperative treatment, however, is not specified or agreed on. It could involve simple activity modification, bracing, or even casting.

Successful treatment of OCD has remained vague in the literature. The only specifics in regard to assessing outcomes have been radiographic and were discussed previously regarding variable definitions of healing. Success is sometimes reported when the clinical symptoms have resolved, but no study to date has defined an outcome measure for OCD lesions. Bearing this in mind, the rates of healing for nonoperative treatment have varied from 50% to 94% [8, 9, 11, 13, 26, 28, 31, 32, 35, 48, 49, 70, 85].

Wall et al. [80] used a standard protocol to nonoperatively treat 42 children with stable OCD lesions. Treatment was initiated with 6 weeks of weightbearing immobilization in a cylinder cast. If the lesion demonstrated reossification on radiography, then casting was discontinued and the patients moved to Phase 2. If reossification was not present, then the children (after a 1-week holiday for ROM) were casted for another 6 weeks. Phase 2 was then started by placing them in an unloader brace and restricting them from running, jumping, and sports. During this phase, children were radiographed every 6 to 8 weeks and activity was slowly advanced as long as radiographs demonstrated progression of healing. At 6 months of nonoperative treatment, 66% of the patients reported no pain.

Surgery is generally indicated for unstable OCD lesions and stable OCD lesions that fail nonoperative treatment [12, 40]. The variability in surgeon preference for surgical treatment is reflected by the variability in surgical methods. These include drilling (both retrograde and antegrade [6, 17, 30, 39, 46], bone grafting [34, 46, 72], fixation [9, 26, 32, 37], alignment procedures [71], and débridement [26]. Most authors have moved toward arthroscopic treatment that often involves small-gauge drilling and the possibility of lesion fixation [12, 17, 30, 39, 46]. In stable lesions undergoing surgery, arthroscopic drilling is a commonly used treatment choice, although the literature is inconclusive of the benefit of this treatment over any other surgical modality [12]. The concept behind drilling is that when the dense rim of the lesion is perforated, the healing response brings about neovascularization and ossification of the cartilaginous lesion as a result of migration of inflammatory factors and stem cells [17]. We emphasize, however, that the pathophysiology of an OCD lesion is not fully understood and therefore treatment modalities are based merely on unproven hypotheses.

Rates of healing (whether defined as radiographic or symptomatic relief) range from 82% to 98% with arthroscopic drilling [5, 7, 30, 39, 45]. Most authors report times to radiographic healing ranging from 6 weeks to 2 years. There is debate regarding the method of drilling: transarticular versus intraepiphyseal. Whereas the intraepiphyseal technique has the advantage of not violating the intact articular cartilage, it comes at the expense of being technically more demanding and requiring fluoroscopy [17]. Lebolt and Wall [45] further describe an intraepiphyseal technique without small-gauge drilling using a larger caliber, cannulated drill bit in which the drill tract is then back-filled with bone graft. This technique, then, is theoretically both osteoconductive and osteoinductive.

If the cartilage is unstable, then drilling can be done directly into the dense rim without violating the overlying loose cartilage. Surgical fixation is then indicated; and, if possible, then the fibrous tissue between the lesion and the condyle should be removed. This curettage is often not possible if the lesion is merely ballotable. Bone grafting should also be considered for these lesions to help restore articular congruity after fixation. Multiple techniques have been described for fixation in OCD. Metallic compression screws have been used with success (radiographic union of the OCD lesion and resolution of symptoms); however, even the authors that promote this technique indicate that midterm (3-year) followup demonstrated an occasional (four of 35) failure [27, 34]. Headed screws may damage opposing articular cartilage and should be removed at a second procedure usually 6 to 12 weeks after implantation, which provides an opportunity to assess healing and stability of the lesion, yet to minimize risk and decrease need

for second surgeries, several authors have advocated the use of headless compression screws with 88% to 100% healing being reported [42, 51, 52, 77]. Screws tend to work best when there is substantial bone remnant in the OCD lesion to provide purchase. Bioabsorbable implants offer some hypothetical advantages related to decreased need for removal and the lack of interference when using MRI to assess healing [73]. Two authors have reported healing rates (seven of seven and seven of eight) and no major complications [44, 61]. Furthermore, authors have demonstrated no adverse reactions resulting from the degradation of modern implants [76]. However, these implants are not without their complications: breakage with loose body formation, opposing cartilage damage, and possible cyst formation and synovitis [22]. Another technique currently used to treat OCD lesions is biologic fixation, meaning the use of osteochondral plugs for fixation. This technique may offer both biologic and mechanical stability. Several authors have reported healing on MRI followup studies with approximately 66% to 95% having good to excellent clinical outcomes using either the Hughston rating or the International Cartilage Repair Society (ICRS) [20, 38, 57]. Miniaci and Tytherleigh-Strong [55] used multiple 4.5-mm osteochondral autograft plugs. At 18-month followup, all 20 patients scored a rating of normal on the International Knee Documentation Committee (IKDC) score and MRI demonstrated incorporation of the osseous portion of the plug at 6 months and the cartilage portion at 9 months.

If the OCD lesion is fragmented or is not amenable to fixation as a result of cartilage quality or incongruence, then the fragment should be removed, the donor site débrided, and the defect addressed based on individual findings [12, 53, 83]. Excision of the fragment may alleviate short-term pain but is associated with only 29% radiographic resolution and 79% with degenerative findings on plain film at a mean 11 years followup [53, 60]. Wright et al. [83] demonstrated that 65% of patients had a fair to poor result on the Hughston radiographic scale (at 8.9-year followup) with excision alone. Therefore, salvage procedures should include an additional intervention beyond fragment excision.

Microfracture is commonly used for traumatic osteochondral defects [4], but its use in OCD is less defined. Traumatic cartilage defects typically have relatively intact subchondral bone in contrast to OCD, in which the subchondral bone is believed to be the primary site of pathology. Therefore, in OCD lesions, the defect created after débridement can be quite deep, leading us to question whether microfracture can adequately restore subchondral support and joint congruity. Gudas et al. [29] reported a prospective, randomized study comparing microfracture with osteochondral autologous transplantation in OCD and found that both groups demonstrated substantial improvement initially in clinical symptoms and in their ICRS scores, but the microfracture group deteriorated over time with 41% failing (based on pain and joint swelling necessitating a second-look surgery) at 4 years compared with none in the transplant group.

Autologous chondrocyte transplantation may be a good choice for large defects with authors reporting between 91% and 96% good to excellent results (based on multiple outcome scores both clinician- and patient-derived such as the Brittberg clinical rating, Cincinnati scoring, and five others and converted in each paper as excellent, good, fair, or poor) at 5 years [4, 29, 66]. Another possible salvage procedure with short-term results of IKDC improvement from 37 to 70 points, although not currently available in the United States, is matrix-supported autogenous chondrocyte implantation [75]. Fresh osteochondral allograft offers the ability to simultaneously address the bone and cartilage defects with a single graft with good pain relief in large lesions [18, 23]. Furthermore, it may be important to consider other associated factors in the treatment of OCD. This includes ligamentous stability and appropriate mechanical axis alignment [23, 25].

Discussion

We sought to address four issues: (1) the etiology of OCD; (2) the presence and locations; (3) the most appropriate diagnostic tests; and (4) the most effective treatment strategies. Unfortunately, after 123 years, the body of literature provides us with little definitive evidence on which to base diagnosis and treatment. This dearth of evidence led to the formation of the ROCK study group. The aim of ROCK is to systematically study the etiology, diagnosis, and treatment of OCD in a multicenter, prospective fashion. The following paragraphs summarize our current understanding of the literature as it relates to the four proposed questions.

This review, and the literature in general, expose major limitations in our knowledge. First, OCD is a relatively rare condition. The low incidence of disease makes it difficult for any single surgeon to treat enough cases to perform any meaningful comparative research. Hence, small, retrospective case series represent the bulk of the literature on OCD. Unfortunately, case series suffer from considerable opportunities for bias and, as a result of substantial variations in methodology, are impossible to compare. Recently, the AAOS Clinical Practice Guideline Committee published the result of its literature review [44]. The initial literature search yielded 1215 articles related to OCD published since 1966. For an article to be considered as evidence on which to base guidelines, each article's

methodology was rigorously scrutinized. Articles on nonhuman subjects, retrospective case series, and in vitro studies were eliminated. This process left only 16 articles that could be considered for the basis of the guidelines. This process exposed the deficiencies in the literature and was a primary motivation for the formation of the ROCK study group. Second, the diagnosis has not been clearly defined in many studies. For example, the definition of healing has ranged from lack of pain to complete radiographic resolution. Few studies include validated, patient-oriented outcome instruments such as IKDC. Even the definition of disease is often unclear. Papers on OCD have included both adults and children and even mixed osteochondritis dissecans with acute, traumatic osteochondral fractures. Third, outcomes are inconsistently reported, making comparison of treatments difficult.

Although no direct cause has been elucidated in the literature, factors such as high levels of activity and frequent participation in sports seem to be the most consistently associated factors in patients with OCD, supporting the repetitive trauma theory. Although the true cause is likely multifactorial, causes such as inflammation, direct genetic inheritance, and ischemia have not been consistently supported by the literature.

The literature does support that most of the subjects diagnosed with OCD present with vague, activity-related pain for long periods of time before a definitive diagnosis. Mechanical symptoms and joint effusion are found in a minority of cases at initial presentation. The most common location for OCD in the knee is the lateral aspect of the medial femoral condyle followed by the lateral femoral condyle and the patellofemoral joint. These locations and ranking of frequency have been reproduced in the literature.

The majority of OCD lesions can be diagnosed on plain radiographs. However, MRI can provide valuable information regarding the integrity of the articular cartilage and the interface between the lesion and the surrounding bone. Currently, these factors are believed to be important when classifying a lesion as stable or unstable.

This is likely the most controversial question. Most authors agree that stable OCD lesions in skeletally immature patients should be treated initially with some form of nonoperative treatment for 3 to 6 months before escalation to operative intervention. However, nonoperative treatment ranges from simple activity modification to casting and there is no definitive evidence to recommend a particular therapy. Stable lesions failing nonoperative treatment can be considered for arthroscopic drilling. Both transarticular and retroarticular techniques have their supporters; however, neither technique has demonstrated a clear advantage in outcome. Unstable lesions have a broad range of options, again with no clear advantage of one technique over another. Options include: fixation with screws, bone grafting, fixation with autograft osteochondral plugs, and salvage procedures such as autologous chondrocyte implantation and fresh osteochondral allografts.

Osteochondritis dissecans is a long recognized yet poorly understood condition. The exact cause and natural history remain elusive in the literature. Although the symptoms of OCD can be vague, plain radiographs can make the diagnoses; MRI can further characterize the lesion's stability. In those cases in which the lesion appears stable, a trial of nonoperative therapy may be attempted for a period of 3 to 6 months. Nonoperative treatment consists of activity restriction with or without immobilization and close radiographic followup. In cases of unstable lesions and those failing nonoperative treatment, surgical treatment is indicated based on the individual situation. Although disheartening to the orthopaedic surgeon, our lack of understanding of OCD represents an opportunity to perform high-quality, prospective, comparative research. This opportunity is the impetus for the formation of the ROCK study group. ROCK will, hopefully, contribute some of the answers to our remaining questions over the next two decades.

References

1. Aichroth P. Osteochondritis dissecans of the knee. A clinical survey. *J Bone Joint Surg Br.* 1971;53:440–447.
2. Barrie HJ. Hypertrophy and laminar calcification of cartilage in loose bodies as probable evidence of an ossification abnormality. *J Pathol.* 1980;132:161–168.
3. Barrie HJ. Hypothesis—a diagram of the form and origin of loose bodies in osteochondritis dissecans. *J Rheumatol.* 1984;11:512–513.
4. Bedi A, Feeley BT, Williams RJ 3rd. Management of articular cartilage defects of the knee. *J Bone Joint Surg Am.* 2010;92:994–1009.
5. Bradley J, Dandy DJ. Osteochondritis dissecans and other lesions of the femoral condyles. *J Bone Joint Surg Br.* 1989;71:518–522.
6. Bradley J, Dandy DJ. Results of drilling osteochondritis dissecans before skeletal maturity. *J Bone Joint Surg Br.* 1989;71:642–644.
7. Bruns J, Rayf M, Steinhagen J. Longitudinal long-term results of surgical treatment in patients with osteochondritis dissecans of the femoral condyles. *Knee Surg Sports Traumatol Arthrosc.* 2008;16:436–441.
8. Cahill B. Treatment of juvenile osteochondritis dissecans and osteochondritis dissecans of the knee. *Clin Sports Med.* 1985;4:367–384.
9. Cahill BR. Osteochondritis dissecans of the knee: treatment of juvenile and adult forms. *J Am Acad Orthop Surg.* 1995;3:237–247.
10. Cahill BR, Ahten SM. The three critical components in the conservative treatment of juvenile osteochondritis dissecans (JOCD). Physician, parent, and child. *Clin Sports Med.* 2001;20:287–298, vi.
11. Cahill BR, Phillips MR, Navarro R. The results of conservative management of juvenile osteochondritis dissecans using joint scintigraphy. A prospective study. *Am J Sports Med.* 1989;17:601–605; discussion 605–606.

12. Chambers HG, Shea KG, Carey JL. AAOS Clinical Practice Guideline: diagnosis and treatment of osteochondritis dissecans. *J Am Acad Orthop Surg.* 2011;19:307–309.
13. Chiroff RT, Cooke CP 3rd. Osteochondritis dissecans: a histologic and microradiographic analysis of surgically excised lesions. *J Trauma.* 1975;15:689–696.
14. Crawford DC, Safran MR. Osteochondritis dissecans of the knee. *J Am Acad Orthop Surg.* 2006;14:90–100.
15. De Smet AA, Fisher DR, Graf BK, Lange RH. Osteochondritis dissecans of the knee: value of MR imaging in determining lesion stability and the presence of articular cartilage defects. *AJR Am J Roentgenol.* 1990;155:549–553.
16. De Smet AA, Ilahi OA, Graf BK. Reassessment of the MR criteria for stability of osteochondritis dissecans in the knee and ankle. *Skeletal Radiol.* 1996;25:159–163.
17. Edmonds EW, Albright J, Bastrom T, Chambers HG. Outcomes of extra-articular, intra-epiphyseal drilling for osteochondritis dissecans of the knee. *J Pediatr Orthop.* 2010;30:870–878.
18. Emmerson BC, Gortz S, Jamali AA, Chung C, Amiel D, Bugbee WD. Fresh osteochondral allografting in the treatment of osteochondritis dissecans of the femoral condyle. *Am J Sports Med.* 2007;35:907–914.
19. Fairbanks H. Osteo-chondritis dissecans. *Br J Surg.* 1933;21:67–82.
20. Fonseca F, Balaco I. Fixation with autogenous osteochondral grafts for the treatment of osteochondritis dissecans (stages III and IV). *Int Orthop.* 2009;33:139–144.
21. Freifberg AH, Woolley PG. Osteochondritis dissecans: concerning its nature and relation to formation of joint mice. *J Bone Joint Surg Am.* 1910; s2–s8: 477–494.
22. Friederichs MG, Greis PE, Burks RT. Pitfalls associated with fixation of osteochondritis dissecans fragments using bioabsorbable screws. *Arthroscopy.* 2001;17:542–545.
23. Garrett JC. 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.
24. Gebarski K, Hernandez RJ. Stage-I osteochondritis dissecans versus normal variants of ossification in the knee in children. *Pediatr Radiol.* 2005;35:880–886.
25. Ghazavi MT, Pritzker KP, Davis AM, Gross AE. Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. *J Bone Joint Surg Br.* 1997;79:1008–1013.
26. Glancy GL. Juvenile osteochondritis dissecans. *Am J Knee Surg.* 1999;12:120–124.
27. Gomoll AH, Flik KR, Hayden JK, Cole BJ, Bush-Joseph CA, Bach BR Jr. Internal fixation of unstable Cahill Type-2C osteochondritis dissecans lesions of the knee in adolescent patients. *Orthopedics.* 2007;30:487–490.
28. Green WT, Banks HH. Osteochondritis dissecans in children. *J Bone Joint Surg Am.* 1953;35:26–47; passim.
29. Gudas R, Simonaityte R, Cekanauskas E, Tamosiunas R. A prospective, randomized clinical study of osteochondral autologous transplantation versus microfracture for the treatment of osteochondritis dissecans in the knee joint in children. *J Pediatr Orthop.* 2009;29:741–748.
30. Guhl JF. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res.* 1982;167:65–74.
31. Hefti F, Beguiristain J, Krauspe R, Moller-Madsen B, Riccio V, Tschauner C, Wetzel R, Zeller R. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B.* 1999;8:231–245.
32. Hughston JC, Hergenroeder PT, Courtenay BG. Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am.* 1984;66:1340–1348.
33. Jacobi M, Wahl P, Bouaicha S, Jakob RP, Gautier E. Association between mechanical axis of the leg and osteochondritis dissecans of the knee: radiographic study on 103 knees. *Am J Sports Med.* 2010;38:1425–1428.
34. Johnson LL, Uitvlugt G, Austin MD, Detrisac DA, Johnson C. Osteochondritis dissecans of the knee: arthroscopic compression screw fixation. *Arthroscopy.* 1990;6:179–189.
35. Jurgensen I, Bachmann G, Schleicher I, Haas H. Arthroscopic versus conservative treatment of osteochondritis dissecans of the knee: value of magnetic resonance imaging in therapy planning and follow-up. *Arthroscopy.* 2002;18:378–386.
36. Kijowski R, Blankenbaker DG, Shinki K, Fine JP, Graf BK, De Smet AA. Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. *Radiology.* 2008;248:571–578.
37. Kivisto R, Pasanen L, Leppilahti J, Jalovaara P. Arthroscopic repair of osteochondritis dissecans of the femoral condyles with metal staple fixation: a report of 28 cases. *Knee Surg Sports Traumatol Arthrosc.* 2002;10:305–309.
38. Kobayashi T, Fujikawa K, Oohashi M. Surgical fixation of massive osteochondritis dissecans lesion using cylindrical osteochondral plugs. *Arthroscopy.* 2004;20:981–986.
39. Kocher MS, Micheli LJ, Yaniv M, Zurakowski D, Ames A, Adrignolo AA. Functional and radiographic outcome of juvenile osteochondritis dissecans of the knee treated with transarticular arthroscopic drilling. *Am J Sports Med.* 2001;29:562–566.
40. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med.* 2006;34:1181–1191.
41. Konig F. Ueber freie korper in den gelenken. *Dtsch Z Chir.* 1887;27:90–109.
42. Kouzelis A, Plessas S, Papadopoulos AX, Gliatis I, Lambiris E. Herbert screw fixation and reverse guided drillings, for treatment of types III and IV osteochondritis dissecans. *Knee Surg Sports Traumatol Arthrosc.* 2006;14:70–75.
43. Kozlowski K, Middleton R. Familial osteochondritis dissecans: a dysplasia of articular cartilage? *Skeletal Radiol.* 1985;13:207–210.
44. Larsen MW, Pietrzak WS, DeLee JC. Fixation of osteochondritis dissecans lesions using poly(L-lactic acid)/poly(glycolic acid) copolymer bioabsorbable screws. *Am J Sports Med.* 2005;33:68–76.
45. Lebolt JR, Wall EJ. Retroarticular drilling and bone grafting of juvenile osteochondritis dissecans of the knee. *Arthroscopy.* 2007;23:794.e1–e4.
46. Lee CK, Mercurio C. Operative treatment of osteochondritis dissecans in situ by retrograde drilling and cancellous bone graft: a preliminary report. *Clin Orthop Relat Res.* 1981;158:129–136.
47. Linden B. The incidence of osteochondritis dissecans in the condyles of the femur. *Acta Orthop Scand.* 1976;47:664–667.
48. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. *J Bone Joint Surg Am.* 1977;59:769–776.
49. Lindholm TS, Osterman K. Treatment of juvenile osteochondritis dissecans in the knee. *Acta Orthop Belg.* 1979;45:633–640.
50. Litchman HM, McCullough RW, Gandsman EJ, Schatz SL. Computerized blood flow analysis for decision making in the treatment of osteochondritis dissecans. *J Pediatr Orthop.* 1988;8:208–212.
51. Mackie IG, Pemberton DJ, Maheson M. Arthroscopic use of the Herbert screw in osteochondritis dissecans. *J Bone Joint Surg Br.* 1990;72:1076.
52. Makino A, Muscolo DL, Puigdevall M, Costa-Paz M, Ayerza M. Arthroscopic fixation of osteochondritis dissecans of the knee: clinical, magnetic resonance imaging, and arthroscopic follow-up. *Am J Sports Med.* 2005;33:1499–1504.
53. Michael JW, Wurth A, Eysel P, Konig DP. Long-term results after operative treatment of osteochondritis dissecans of the knee joint-30 year results. *Int Orthop.* 2008;32:217–221.

54. Milgram JW. Radiological and pathological manifestations of osteochondritis dissecans of the distal femur. A study of 50 cases. *Radiology.* 1978;126:305–311.
55. Miniaci A, Tytherleigh-Strong G. Fixation of unstable osteochondritis dissecans lesions of the knee using arthroscopic autogenous osteochondral grafting (mosaicplasty). *Arthroscopy.* 2007;23:845–851.
56. Mitsuoka T, Shino K, Hamada M, Horibe S. Osteochondritis dissecans of the lateral femoral condyle of the knee joint. *Arthroscopy.* 1999;15:20–26.
57. Miura K, Ishibashi Y, Tsuda E, Sato H, Toh S. Results of arthroscopic fixation of osteochondritis dissecans lesion of the knee with cylindrical autogenous osteochondral plugs. *Am J Sports Med.* 2007;35:216–222.
58. Mubarak SJ, Carroll NC. Familial osteochondritis dissecans of the knee. *Clin Orthop Relat Res.* 1979;140:131–136.
59. Mubarak SJ, Carroll NC. Juvenile osteochondritis dissecans of the knee: etiology. *Clin Orthop Relat Res.* 1981;157:200–211.
60. Murray JR, Chitnavis J, Dixon P, Hogan NA, Parker G, Parish EN, Cross MJ. Osteochondritis dissecans of the knee; long-term clinical outcome following arthroscopic débridement. *Knee.* 2007;14:94–98.
61. Nakagawa T, Kurosawa H, Ikeda H, Nozawa M, Kawakami A. Internal fixation for osteochondritis dissecans of the knee. *Knee Surg Sports Traumatol Arthrosc.* 2005;13:317–322.
62. Nawata K, Teshima R, Morio Y, Hagino H. Anomalies of ossification in the posterolateral femoral condyle: assessment by MRI. *Pediatr Radiol.* 1999;29:781–784.
63. O'Connor MA, Palaniappan M, Khan N, Bruce CE. Osteochondritis dissecans of the knee in children. A comparison of MRI and arthroscopic findings. *J Bone Joint Surg Br.* 2002;84:258–262.
64. Paget J. On the production of some of the loose bodies in joints. *St Bartholomew's Hospital Reports.* 1870;6:1–4.
65. Paletta GA Jr, Bednarz PA, Stanitski CL, Sandman GA, Stanitski DF, Kottamasu S. The prognostic value of quantitative bone scan in knee osteochondritis dissecans. A preliminary experience. *Am J Sports Med.* 1998;26:7–14.
66. Peterson L, Minas T, Brittberg M, Lindahl A. 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.
67. Petrie PW. Aetiology of osteochondritis dissecans. Failure to establish a familial background. *J Bone Joint Surg Br.* 1977;59: 366–367.
68. Phillips HO, Grubb SA. Familial multiple osteochondritis dissecans. Report of a kindred. *J Bone Joint Surg Am.* 1985;67:155–156.
69. Ribbing S. Studies on hereditary, multiple epiphyseal disorder. *Acta Radiol.* 1937;34:1–107.
70. Schenck RC Jr, Goodnight JM. Osteochondritis dissecans. *J Bone Joint Surg Am.* 1996;78:439–456.
71. Slawski DP. High tibial osteotomy in the treatment of adult osteochondritis dissecans. *Clin Orthop Relat Res.* 1997;341:155–161.
72. Slough JA, Noto AM, Schmidt TL. Tibial cortical bone peg fixation in osteochondritis dissecans of the knee. *Clin Orthop Relat Res.* 1991;267:122–127.
73. Smith DS, Sharp DC, Resendes M. MRI of healing osteochondritis dissecans fragment with absorbable pins. *J Comput Assist Tomogr.* 1994;18:832–833.
74. Stanitski CL, Bee J. Juvenile osteochondritis dissecans of the lateral femoral condyle after lateral discoid meniscal surgery. *Am J Sports Med.* 2004;32:797–801.
75. Steinhagen J, Bruns J, Deuretzbacher G, Ruether W, Fuerst M, Niggemeyer O. Treatment of osteochondritis dissecans of the femoral condyle with autologous bone grafts and matrix-supported autologous chondrocytes. *Int Orthop.* 2010;34:819–825.
76. Tabaddor RR, Banffy MB, Andersen JS, McFeely E, Ogunwole O, Micheli LJ, Kocher MS. Fixation of juvenile osteochondritis dissecans lesions of the knee using poly 96L/4D-lactide copolymer bioabsorbable implants. *J Pediatr Orthop.* 2010;30:14–20.
77. Thomson NL. Osteochondritis dissecans and osteochondral fragments managed by Herbert compression screw fixation. *Clin Orthop Relat Res.* 1987;224:71–78.
78. Uozumi H, Sugita T, Aizawa T, Takahashi A, Ohnuma M, Itoi E. Histologic findings and possible causes of osteochondritis dissecans of the knee. *Am J Sports Med.* 2009;37:2003–2008.
79. Wall E, Von Stein D. Juvenile osteochondritis dissecans. *Orthop Clin North Am.* 2003;34:341–353.
80. Wall EJ, Vourazeris J, Myer GD, Emery KH, Divine JG, Nick TG, Hewett TE. The healing potential of stable juvenile osteochondritis dissecans knee lesions. *J Bone Joint Surg Am.* 2008;90: 2655–2664.
81. Wilson JN. A diagnostic sign in osteochondritis dissecans of the knee. *J Bone Joint Surg Am.* 1967;49:477–480.
82. Woods K, Harris I. Osteochondritis dissecans of the talus in identical twins. *J Bone Joint Surg Br.* 1995;77:331.
83. Wright RW, McLean M, Matava MJ, Shively RA. Osteochondritis dissecans of the knee: long-term results of excision of the fragment. *Clin Orthop Relat Res.* 2004;424:239–243.
84. Yonetani Y, Nakamura N, Natsuume T, Shiozaki Y, Tanaka Y, Horibe S. Histological evaluation of juvenile osteochondritis dissecans of the knee: a case series. *Knee Surg Sports Traumatol Arthrosc.* 2010;18:723–730.
85. Yoshida S, Ikata T, Takai H, Kashiwaguchi S, Katoh S, Takeda Y. Osteochondritis dissecans of the femoral condyle in the growth stage. *Clin Orthop Relat Res.* 1998;346:162–170.