Osteochondritis Dissecans of the Knee: Treatment of Juvenile and Adult Forms

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Abstract

Osteochondritis dissecans (OCD) and juvenile osteochondritis dissecans (JOCD) are distinct entities that require different management. Although both conditions result from stress fractures of the subchondral bone, JOCD has a much better prognosis; treated conservatively, 50% of cases will heal, probably providing a normal knee during adult life. In contrast, OCD often is followed by the early onset of degenerative arthritis. The treatment of JOCD and OCD, whether nonoperative or operative, should be based on the principles of fracture treatment. Unfortunately, surgical correction of either of these conditions is unlikely to succeed unless the joint surface is perfectly restored.

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Osteochondritis dissecans (OCD) and juvenile osteochondritis dissecans (JOCD) present a clinical conundrum to many orthopaedic surgeons. There is substantial debate regarding the etiology and incidence of both conditions. There is also uncertainty about the adult consequences of JOCD and the indications for surgery and long-term surgical results in patients with JOCD.

Some of the confusion arises because these two quite different entities are often presented as the same condition in scientific publications. The important distinguishing feature is that JOCD occurs in patients with an open epiphyseal plate (physis). Once established, OCD has a significantly poorer prognosis than JOCD and rarely heals or unites without operative intervention. This differential diagnosis is the principal clinical distinguishing factor between the two osteochondritides. The overlap between the two occurs when the lesion does not heal before closure of the physis of the distal femur. Although the exact pathophysiology of this prognostic difference is unknown, it is temporally related to physeal closure and may be due to the concomitant vascular alteration.

It has also been questioned whether OCD can begin *de novo* in the distal femur. In my own series of patients, there seems to be little question that this condition can arise *de novo* in the skeletally mature knee. I have seven patients with OCD, all male, who had no history of previous knee symptoms and had normal radiographs (anteroposterior, lateral, and tunnel views) of diagnostic quality showing an open physis before the onset of adult symptoms of OCD. Their age at presentation ranged from 17 to 36 years. The majority of my younger OCD patients have a history of knee symptoms dating back to a time when their physes would have been open; these cases probably represent JOCD that did not heal and evolved to OCD.

The true incidence of *de novo* OCD is not known (Fig. 1). Most older patients with OCD either have uncertain histories of prior knee symptoms or no previous radiographs obtained when their physes were open. It is believed that OCD can begin at any time up to age 50, suggesting that older patients do represent *de novo* cases. I have concluded that although older patients may have true adult-onset OCD, most OCD cases, especially in younger adults, probably arise from established JOCD.

Etiology

Since König's coining of the term "osteochondritis dissecans" in 1887,[1] there has been a proliferation of publications dealing with its etiology and pathology. Most of the early publications were abrasively critical of König's new terminology, and from this he soon learned that his appellation for OCD was inappropriate. According to Barrie,[2] "Axhausen in 1924 attempted to replace the

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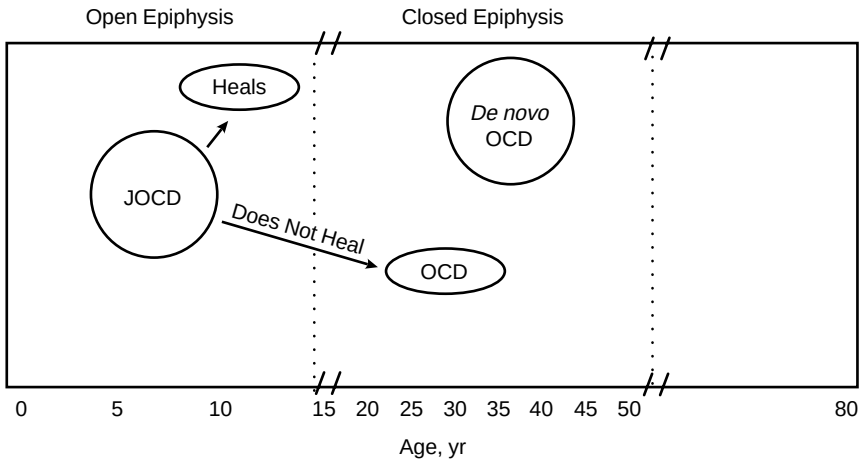

Fig. 1 The age ranges at which JOCD and OCD occur. Juvenile OCD begins before closure of the epiphyseal plate. Approximately half of these lesions will heal with conservative treatment. If the lesion does not heal before physeal closure, OCD results. While *de novo* OCD is known to occur, its incidence is unknown.

name with 'Abgrenzungsvorgang' but this [abridgment] was doomed from the start."

The combining of JOCD and OCD in these early publications has led to confusion regarding the etiology, treatment, and prognosis of these conditions, because of the mixed clinical material. While there is some agreement that JOCD represents the result of cumulative stress to subchondral bone, resulting in subchondral stress fractures, other possible causes are poorly supported by scientific evidence. Both conditions have been associated with endocrinopathies, familial conditions, accessory centers of ossification, osteochondral fractures, osteonecrosis, and even carpal tunnel syndrome.

In my 204 patients with JOCD, most reported no obvious single traumatic event. What is common to most of these cases is a long history of exercise or sports. This observation is similar to the findings of both Lindén[3] and Aichroth,[4] who cited exposure to sports at a young age as an important etiologic factor in JOCD. For this reason, I believe that an emphasis on exercise for our youth may be the principal etiologic agent in JOCD.

Other authors have reported that JOCD is associated with many other musculoskeletal abnormalities. Among these are ligamentous laxity, genu varum, genu valgum, patellar malalignment, Sinding-Larsen-Johansson disease, and Osgood-Schlatter disease. Many of these conditions may contribute to stress accumulation on the femoral condyles, which could provoke subchondral injuries.

It is generally recognized that the target tissue in JOCD is the subchondral bone of the distal femoral condyle, rather than the articular cartilage. Stress fractures may develop in subchondral tissue subjected to cyclic, cumulative stresses. This subchondral injury may then provoke vascular compromise, resulting in the typical JOCD lesion of a variably viable bone nucleus covered with intact articular cartilage.

The etiology of JOCD and OCD must be borne in mind when selecting treatment. Since both conditions result from stress fractures, it is appropriate that their management, whether nonoperative or operative, should be based on the principles of fracture treatment. Smillie[5] stated in 1957 that "it is axiomatic that treatment [should] be related to cause" and therefore that the JOCD lesion should be "regarded [as] a fracture in order that the principles of fracture treatment can be applied."

Epidemiology

In a literature search in 1953 Green and Banks[6] could find only 9 JOCD cases reported. In 1989, in a review of publications that limited patients to those with JOCD, I found only four publications analyzing 163 patients.[7]

Three publications shed some light on the epidemiology of JOCD. In 1947, Lavner[8] reported a prevalence of 4% on knee radiographs taken in a hospital. In 1962, Aegerter and Kirkpatrick,[9] without real evidence, concluded that JOCD "is a rather common orthopedic condition." The most comprehensive study was that reported by Lindén[3] in 1976. He conducted a 10-year review in Malmö, Sweden, in which all knee radiographs at all institutions were examined for OCD and JOCD. In the JOCD age group, Lindén found the prevalence to be 18 / 100,000 in females and 29 / 100,000 in males. Of significance was his observation that during the last 5 years of the study the incidence appeared to be increasing, which he attributed to the increasing popularity of sports in Malmö at that time.

In my practice, the mean age of JOCD patients has decreased. In our first report of JOCD patients in 1983, the average age was 12.9 years. In 1992, the mean age had decreased to 11.3 years.

In that same clinical experience, there is also some suggestion that females are more commonly involved. In our earlier JOCD study,

11% of the patients were female, whereas they now constitute 15% of the JOCD population.

I believe it is possible that the incidence of JOCD has increased because these children are introduced to organized sports at younger and younger ages, and cumulative exercise is increasing annually due to the demands of competition. In addition, most sports now require intensive resistance training for the lower extremities, which theoretically might add to the cumulative exercise dose.

Classification

Anatomic

Anatomic-location classifications of JOCD have been published by numerous authors, including Aichroth,[4] Clanton and DeLee,[10] Cahill and Berg,[11] and Hughston et al[12] (Fig. 2). Although these descriptions are different, they all recognize the need for accurately mapping the location of the lesion and ascertaining its dimensions. The importance of these classifications is to draw attention to the fact that JOCD is a failure of a stress-bearing area of the knee and not a condition afflicting the non-weight-bearing lateral side of the medial femoral condyle. It is not sufficient to record that the lesion is on the lateral femoral condyle; a more precise description of the location and size of the lesion is required. This preciseness, whatever classification is used, could increase the understanding of the prognosis of JOCD, which is affected by both location and size.

Surgical

A surgical classification of JOCD lesions that has been developed can be helpful in describing the lesions (Table 1, Fig. 3). This classification is based on visual and palpable observations made at surgery.

Scintigraphic

In 1983 we classified JOCD on the basis of the appearance on technetium-99m phosphate-compound joint scintigraphy.[11] This biologic classification is based on the degree of activity of the femoral condyles and the adjacent tibial plateau (Figs. 4 and 5). Scintigraphic activity is directly related to the volume of area blood flow and/or osteoblastic activity. This classification recognizes four stages based on scintigraphic activity:

Stage 0 is a normal radiographic and scintigraphic appearance (Fig. 5, A).

Stage I is characterized by normal scintigraphic activity although a defect is visible radiographically on the femoral condyle (Fig. 5, B). This finding may represent a healed lesion or an accessory center of ossification. The normal scintigraphic studies of stage I lesions have usually been prompted by incidental radiographic findings of a condylar defect in a knee that is symptomatic for reasons other than JOCD.

A stage II scan (Fig. 5, C) depicts increased uptake. The lesion is due to JOCD and does not represent an accessory center of ossification. Stage II lesions may be asymptomatic.

Stage III scans demonstrate increased isotope uptake in both the lesion and the femoral condyle (Fig. 5, D).

Stage IV scans depict increased uptake in the adjacent tibial plateau

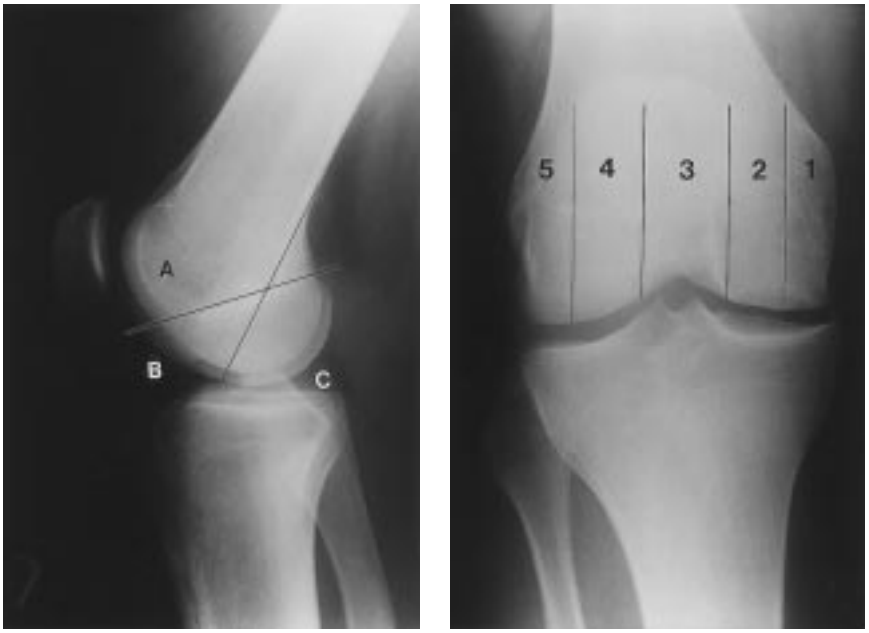

Fig. 2 Anatomic locations of JOCD and OCD in the knee. **A,** Lateral radiograph of a 17-year-old boy with a BC lesion in the medial femoral condyle. Areas B and C are divided by a line projected from the posterior femoral cortex. **B,** Anteroposterior radiograph of the same patient shows a 1-2 lesion occupying the entire weight-bearing area of the femoral condyle. Numbering of the five anatomic areas begins on the medial side. The condyles are bisected, and area 3 is bounded by the walls of the intercondylar notch. (Reproduced with permission from 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;17:601-606.)

Table 1
Classification of JOCD by Appearance of Articular Cartilage and Lesion Mobility at Surgery

Intact cartilage
Stable lesion
Unstable lesion
Disrupted cartilage
Stable lesion
Unstable lesion
Predetachment (no fragment mismatch in acute or chronic cases)
Hinged (fragment mismatch in chronic but not in acute cases)
Loose body (fragment mismatch in chronic but not in acute cases)
Macerated cartilage
Discolored
Blistered
Abraded
Fragmented

(Fig. 5, E), suggesting a response to stress transfer across the joint. If the JOCD lesion is treated successfully, the tibial activity usually decreases and then ceases.

Stages III and IV are always symptomatic. Unless there is compelling clinical or radiographic evidence to the contrary, it can be assumed that the symptoms of patients with stage III and IV lesions are due to pathologic changes caused by JOCD.

Given the temporal insensitivity of radiographs in detecting changes in JOCD activity, joint scintigraphy remains the method of choice for following the progress of the disease. This method exposes the patient to minimal radiation and will reveal changes in lesion activity at 6- to 8-week intervals. It is not necessary to examine JOCD / OCD patients scintigraphically this frequently; however, clinical examinations should be conducted at 8-week intervals. Rescanning at 4-month intervals is adequate.

Parallel-hole scans are obtained only during the first imaging procedure. Thereafter, the much more sensitive and detailed pin-hole technique is used. This is an essential feature of JOCD scintigraphy.

While the scintigraphic technique described here has not provided prognostic guidance, the work of Litchman et al[13] may. They have used computerized blood-flow analysis to decide which JOCD / OCD lesions will heal without surgery and which will not. They have reported their results in 13 patients, only 1 of whom probably had JOCD, and their conclusions seem promising. More work needs to be done in this area.

Conservative Treatment

The history of the treatment of JOCD has been confusing, and there is little consensus. Opinions range from a "do nothing" philosophy to a policy of operating in all cases. Between these extremes are aggressive conservative treatment, such as use of a long-leg cast and nonweight-bearing for periods of up to 18 months. Regardless of the treatment employed, the results are inconclusive because JOCD and OCD cases are mixed, the numbers are small, and there are no controls.

In spite of the confusing data, there is a definite role for nonoperative treatment. The single goal of conservative treatment is to obtain lesion healing before physis closure as a means of preventing early-onset gonarthrosis. Provided the patient is compliant, has a stable lesion, and is not near the age of physis closure, the likelihood is approximately 50% that the lesion will heal within 10 to 18 months.[7] The fact that only half of the cases will have successful outcomes as assessed by radiographic healing does not make an argument for initial operative treatment.

In my JOCD patients treated nonoperatively and followed up for 8 years or longer, normal clinical function and normal radiographs were a

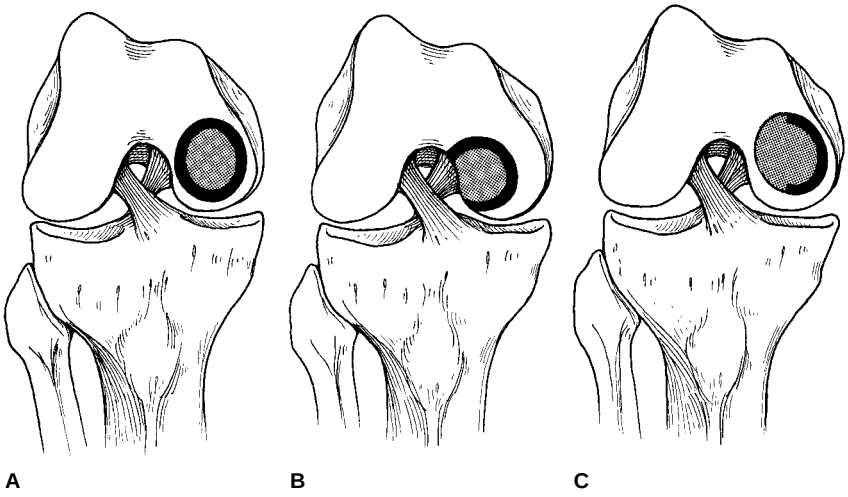

Fig. 3 Classification of JOCD by the appearance of the articular cartilage and the mobility of the lesion. **A,** Predetachment lesion. Note complete circumscription of the lesion. Even though these lesions may seem stable to probing, they should be stabilized. **B,** A hinged lesion of the medial femoral condyle is a frequent finding in this location. The hinge is a soft-tissue vascular bridge from the insertion of the posterior cruciate ligament and should be preserved. **C,** Partially circumscribed lesion. Such lesions may be stable.

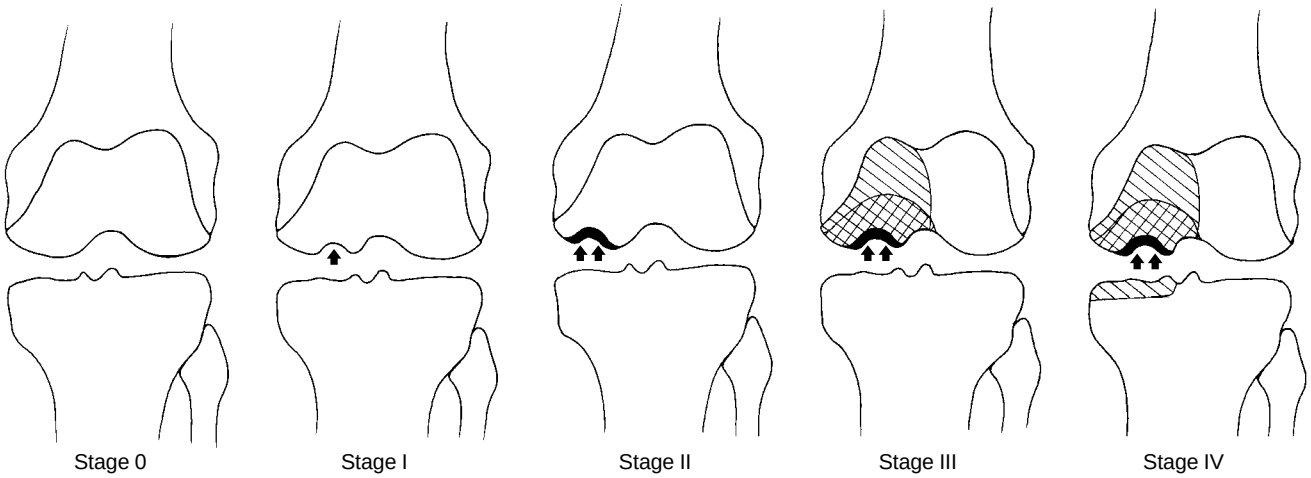

Fig. 4 Classification of JOCD on the basis of the radiographic and scintigraphic appearance. Stage 0 is a normal radiographic and scintigraphic appearance. In stage I, a defect on the femoral condyle is visible radiographically but not scintigraphically. In stage II, the scintigram shows increased uptake in the JOCD lesion. In stage III, increased isotope uptake is seen in both the lesion and the femoral condyle. In stage IV, there is also increased uptake in the adjacent tibial plateau.

striking finding. On review of these patients, I have the impression that conservative treatment of JOCD can be successful and will provide the patient with knee joint function at least as good as, if not better than, that provided by surgical treatment. Even if the patient is within 6 to 12 months of physis closure, a trial of nonoperative treatment is warranted.

General Principles

The basic principle of nonoperative treatment is reduction of exercise to a level where symptom-free activities of daily living are possible.

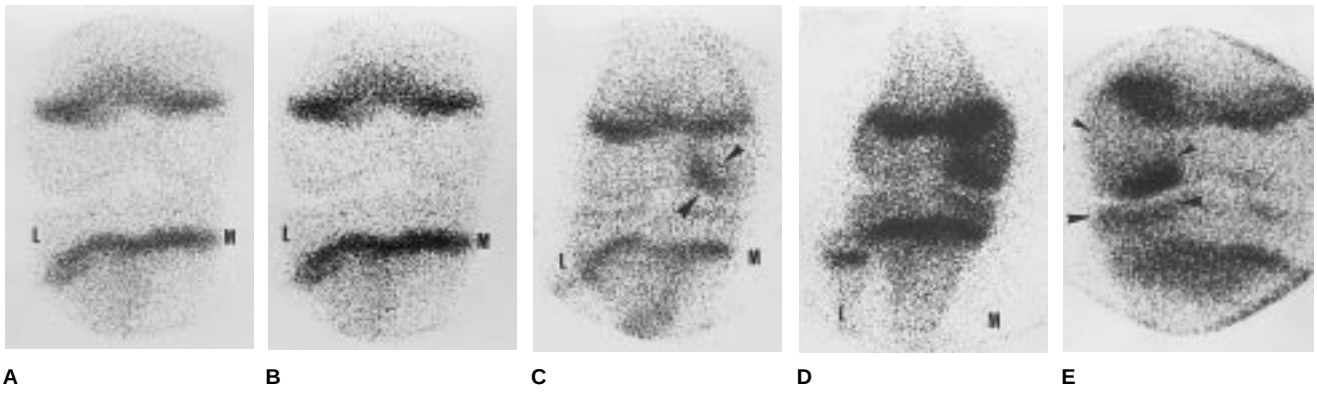

Fig. 5 Scintigraphic appearance of JOCD (L indicates lateral aspect; M, medial aspect). In this scintigraphic classification, stages II, III and IV are additive. **A,** Stage 0. Note the normal activity of the physeal plate. **B,** Stage I scans are scintigraphically normal. This particular scan could be depicting an accessory center of ossification or a healed JOCD defect. **C,** Stage II scans show the first degree of scintigraphic abnormality. On this scan, the medial femoral condyle has a JOCD lesion (arrowheads) of mid- to low-level scintigraphic activity. If this were a preoperative scan, a bone graft should be part of the procedure, since the degree of vascular activity indicated by the scan is marginal. **D,** In this stage III scan, not only the lesion but the entire femoral condyle shows increased uptake. **E,** Stage IV scans exhibit the highest degree of scintigraphic activity. Note the increased uptake in the adjacent tibial plateau, which is likely due to the same process that produces the femoral lesions, namely, increased cumulative stress transfer causing tibial subchondral stress fractures. The intense lesion uptake (arrowheads) indicates that bone grafting to increase vascularity would not be necessary. (Reprinted with permission from Cahill BR, Berg BC: 99m-Technetium phosphate compound joint scintigraphy in the management of juvenile osteochondritis dissecans of the femoral condyles. *Am J Sports Med* 1983;11:329-335.)

This activity modification initially may require the use of crutches for 6 to 8 weeks. Casts, braces, and prolonged non-weight-bearing are not used in my practice. Once the child has reached the symptom-free level, crutches (if used) are discarded, and the patient is followed up with a limited scintigram of the knee and a clinical evaluation every 8 weeks until the clinical and scintigraphic examinations demonstrate that healing has occurred. During this time, competitive sports are eliminated. As healing progresses, recreational cycling, swimming, and lower-extremity strength training are added, provided the child remains asymptomatic.

Patient Compliance

The vast majority of JOCD patients are children of parents who are themselves highly competitive. To suggest conservative treatment that may last 1 year and will eliminate competitive sports for that time provokes astonishment and disbelief in the family.

Parents often feel guilty about having waited 8 to 15 months while symptoms worsened (the usual case presentation) before seeking medical attention. There is also a demand for an immediate return to normal function, usually by surgical methods. If the lesion is not detached, the parents are reassured that the child's history is usual rather than exceptional. Despite their delay in seeking medical attention, the prognosis is not adversely affected, and good results can be expected if nonoperative treatment is selected.

I also use two other arguments against immediate surgical intervention. First, I point out the uncertainty of long-term surgical results when compared with the excellent results of successful nonoperative treatment, provided that the lesion heals before physeal closure. Second, nonoperative and surgical treatments require similar time restrictions on sports participation. This discourse with the parents who seek a quick fix is usually effective in gaining their support to embark on nonoperative treatment.

The family is given educational material on JOCD and is introduced to a support group of previous JOCD families. Since it is unusual for the child to return for the first follow-up visit much improved, further counseling and education are necessary to maintain the family's confidence and compliance with nonoperative treatment.

Follow-up

Since radiography of the knee is relatively insensitive to improvement or worsening of JOCD lesions, joint scintigraphy is the principal tool that I use to measure progression to healing or regression. In my practice, 85% of JOCD patients present initially with a symptomatic knee and a stage III or IV scintigram. Scans should be obtained at 4-month intervals. After a symptom-free level has been attained, scintigraphic activity will decrease slowly; by 6 months the scintigraphic appearance should have improved by at least one stage. Healing is presumed to have occurred when the scintigram depicts a decrease in activity to a low-level stage II, with the scintigraphic activity confined to the lesion. The average time to reach this degree of healing is 10 months. Low-level activity will persist for 6 to 12 months after a return to asymptomatic full activity.

If the patient has a normal clinical examination and a low-level stage II scintigram, return to competitive sports is allowed, provided the symptoms do not recur. A few JOCD patients do not tolerate this increased exercise regimen; the knee becomes symptomatic, and the scintigraphic stage of activity again increases. These patients often respond if sport participation is suspended for another 2 to 3 months.

Operative Treatment

Indications

When should conservative treatment be abandoned? I have used the following criteria[7]: (1) detachment or instability of the fragment while the patient is under treatment; (2) persistence of symptoms in a compliant patient; (3) persistently elevated or worsening scintigraphic activity; and (4) approaching epiphyseal closure. Criterion 1 is an absolute indication, as is a combination of criteria 2, 3, and 4.

Detachment has occurred in 34% of our conservatively treated JOCD patients and has been the most common reason for surgical intervention. Persistent or increasing symptoms and combined indications have each contributed 26% of the failures. The overall success rate of nonoperative treatment remains at 50%.

I have only limited experience with magnetic-pulse bone stimulation. Since there is no information available on the effects of this modality on the epiphyseal plate, I have ceased using it.

Treatment Planning

Figure 6 provides an outline for surgical planning and postoperative care. This algorithm has been developed as an aid in predicting which methods and materials may be necessary to adequately restore the joint surface of the patient with JOCD or OCD. With the exception of earlier surgical intervention, the principles of surgical treatment of OCD are identical to those of JOCD. The use of allografts is also applicable to the OCD patient.

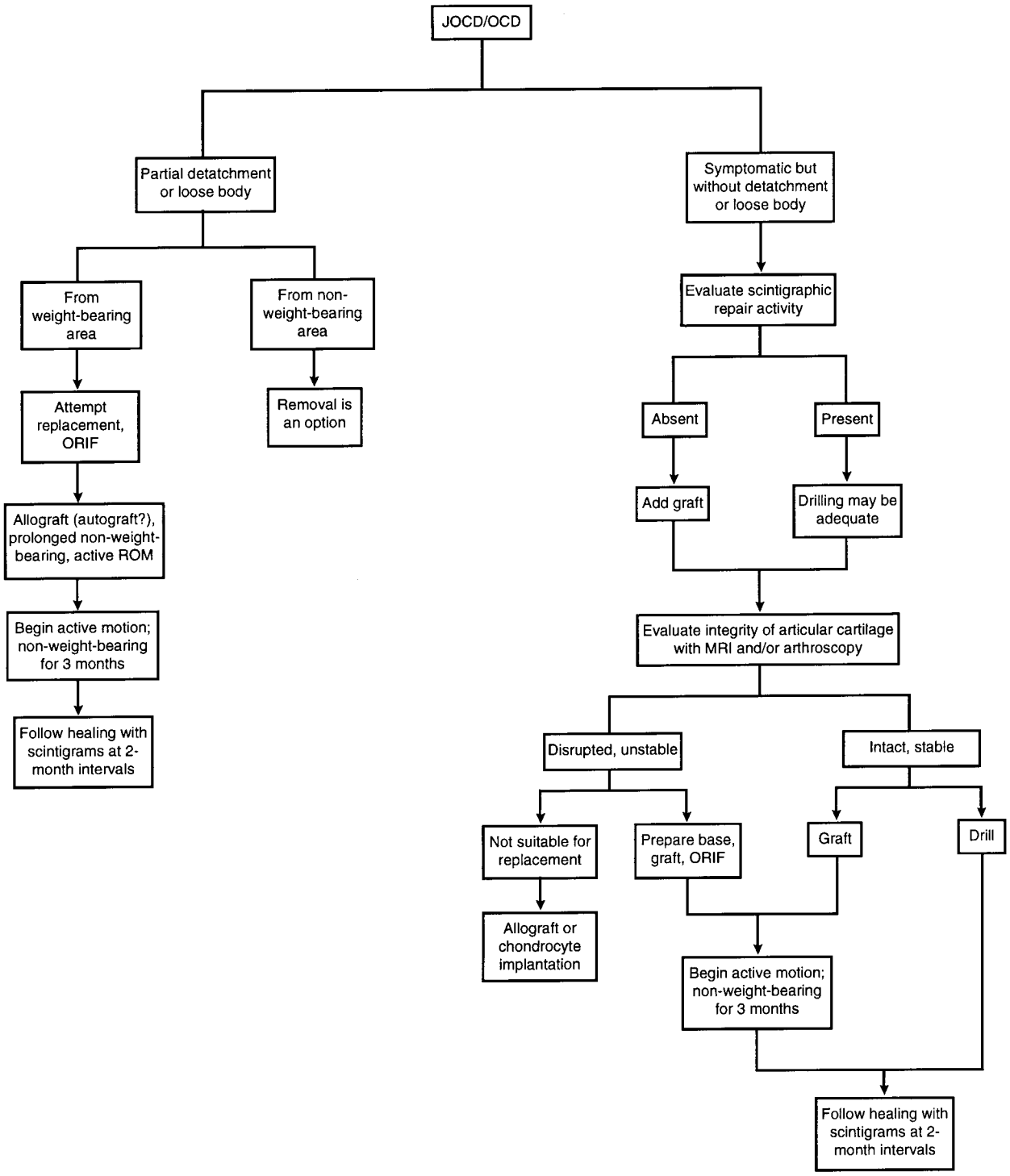

Fig. 6 Algorithm for surgical treatment of JOCD / OCD and postoperative care. Surgical goals are (1) to reestablish the joint surface, (2) to improve the blood supply of the fragment, and (3) to achieve rigid fixation and early motion. MRI = magnetic resonance imaging; ORIF = open reduction and internal fixation; ROM = range-of-motion exercises. (Modified with permission from Cahill B: Treatment of juvenile osteochondritis dissecans and osteochondritis dissecans of the knee. *Clin Sports Med* 1985;4:367-384.)

Loose Body or Partial Detachment

The presence of a loose body or an unstable fragment is an indication for surgery. The diagnosis of an unstable lesion is made on the basis of the history, the clinical examination, and the radiographs. The patient with an unstable fragment (partial detachment) presents with symptoms of knee pain, catching, or locking, and physical examination reveals an effusion. Joint scintigraphy is not helpful in diagnosing lesion instability; however, magnetic resonance (MR) imaging and contrast-enhanced MR imaging may be helpful adjuncts if the symptoms suggest partial detachment but the radiographs are uncertain.

If the lesion is from a weight-bearing area, it is imperative that it be replaced. If the lesion has been unstable or loose for months, the likelihood of restoring the joint surface arthroscopically is small even in the best of hands. The poor results following fragment removal in a weight-bearing area of the femoral condyles are well known, and I believe that this is not a viable option. On rare occasions, a JOCD lesion is so macerated that internal fixation is not possible. At other times, the articular cartilage of the lesion has been destroyed. In both situations, the lesion should be removed and allografting or autografting should be considered, either at the time of lesion removal or at a later date.

Internal fixation is required for loose bodies and unstable or partially detached lesions. Postoperatively, range-of-motion exercises are started at once, but non-weight-bearing is necessary for 3 months.

Failed Nonoperative Treatment of JOCD

When nonoperative treatment fails, preoperative planning is based on the stability of the lesion and its vascular status. If the scintigraphic repair activity is graded stage III or high stage II and the lesion is stable, drilling of the lesion is all that is necessary. If the scintigraphic activity is graded a low stage II or lower, a bone graft should be used to enhance the vascularity of the lesion.

Preoperatively, the integrity of the articular cartilage may be evaluated with MR imaging. Since I perform arthroscopy on all patients, I consider MR imaging unnecessary.

If the lesion is found at arthroscopy to be unstable or is partially detached, it is treated by curettage of the femoral defect, drilling, and internal fixation. Grafting may also be necessary to increase vascularity or to fill subchondral cavity defects. An early decision must be made on whether all of this procedure can be done arthroscopically. If there is any doubt, one should resort to open methods.

If the lesion is stable and the degree of vascularity as demonstrated by scintigraphy is adequate, drilling alone will suffice. This is true whether there is an articular defect partially outlining the JOCD lesion or there is intact cartilage. Usually, if the entire lesion is outlined by a full-thickness cartilage defect, there will also be fragment instability.

If drilling is the only procedure on a stable lesion, the patient is allowed to progress to weight-bearing as tolerated. Most cases are asymptomatic, and the patient can proceed to full weight-bearing within 2 weeks, with follow-up at 2-month intervals until healing occurs.

Growth of the Osteochondritic Fragment

Numerous authors have commented on the growth of partially detached or loose JOCD fragments while the crater of the JOCD lesion in the femoral condyle retains its original dimensions. This creates a fragment mismatch, which makes it difficult to perfectly fit the crater and restore joint-surface orthopticity. This is particularly important when the reduction is attempted arthroscopically. These cases should usually be treated by open methods, since the technical problems posed by arthroscopy are formidable.

After arthrotomy, the lesion is meticulously reduced in size with multiple trial reductions until the fragment exactly fits the crater. When the lesion has been partially detached or loose for months, a sclerotic rim of bone is common. In this circumstance, curetting the crater base to bleeding bone and drilling are imperative. Similarly, the subchondral bone of the fragment is usually sclerotic and also requires curettage. This produces a more extensive depth defect, which will require cancellous grafting from the adjacent nonarticular femoral condyle to obtain restoration of the joint surface. It is recommended that the cancellous grafting should leave the surface 1 to 2 mm high, because there will be some subsidence with healing.

Each trial reduction should provide the surgeon with a best estimate of the proper rotational position of the fragment. When the optimal fragment rotation has been determined, a reference line should be drawn on the fragment and the rim of the crater with methylene blue to ensure that later fixation occurs in the correct position. When dealing with partially detached lesions, every effort should be made to preserve the undetached hinge. This is especially true when the lesion is in area 2-AB (Figs. 2 and 3, B). Lesions in area 2-AB are usually hinged on the soft-tissue bridge of the insertion of the posterior cruciate ligament on the femoral condyle. This soft-tissue hinge should be viewed as a source of some blood supply to the fragment.

Open Versus Arthroscopic Surgery

Before proceeding with definitive operative treatment, I first perform arthroscopy to assess whether an attempt at arthroscopic treatment is feasible. In some cases, preoperative planning will provide several indications that the lesion is not treatable arthroscopically. Examples are lesions that have been detached or unstable for long periods of time, for which the risk of fragment mismatch is high, and lesions located in the A and C areas of the condyle (Fig. 7). Arthroscopically treatable lesions are most commonly located near the AB area of the condyle and have been acutely detached, either wholly or partially.

Arthroscopic treatment should be abandoned when the entire lesion cannot be visualized, when there is inability to determine fragment rotation, or when perfect restoration of joint orthopticity cannot be attained. Miller[14] and Sisk[15] offer the same advice and describe technical details of the surgical management of JOCD and OCD.

In my 124 cases of JOCD treated surgically, open methods were used in 48%. Most cases required extensive fragment remodeling and bone grafting or involved lesions in the A and C areas of the condyle. The remainder were managed arthroscopically.

Fragment Removal

Many clinicians believe that a partially detached lesion and loose bodies that have existed for some time are not suitable for replacement.[14-16] In spite of those opinions, it must be emphasized that removal of potentially reimplantable loose bodies and partially detached lesions that arose from weight-bearing surfaces is to be avoided. The results of removal in long-term follow-up have been poor. The tissue that fills the JOCD crater after treatment is not type I articular cartilage and has a decreased ability to withstand weight-bearing stresses. Conversely, removal of loose bodies from the non-weight-bearing areas, such as the usual site on the medial femoral condyle, will provide good long-term results.

These weight-bearing loose bodies should be treated as described in the previous section on lesion growth and mismatch. The best time to treat these condylar defects is at the first procedure. Although unproved, use of the patient's own tissue would be expected to yield a better long-term survival than the 60% rates reported for allografts.[17,18]

Internal Fixation

The open and arthroscopic techniques of stabilizing JOCD lesions are well described by Sisk[15] and Miller.[14] Selection of the optimal device for stabilization is still being debated, however. The options for internal fixation for JOCD include bone pegs, 0.062-inch-diameter pins, biodegradable pins, and cannulated screws. The goals of internal fixation are to obtain rigid internal fixation that permits early joint motion, to anatomically restore the joint surface, and, if possible, to enhance revascularization of the fragment.

There is no question that cannulated screws will provide rigid fixation, allow early joint motion, and can be relatively easily inserted with arthroscopy. However, the price we pay for these advantages may be offset by the disadvantages. During the past 4 years I have used cannulated screws. When the lesions have been reexamined at the time of fixation removal or second-look arthroscopy, I have been concerned about the

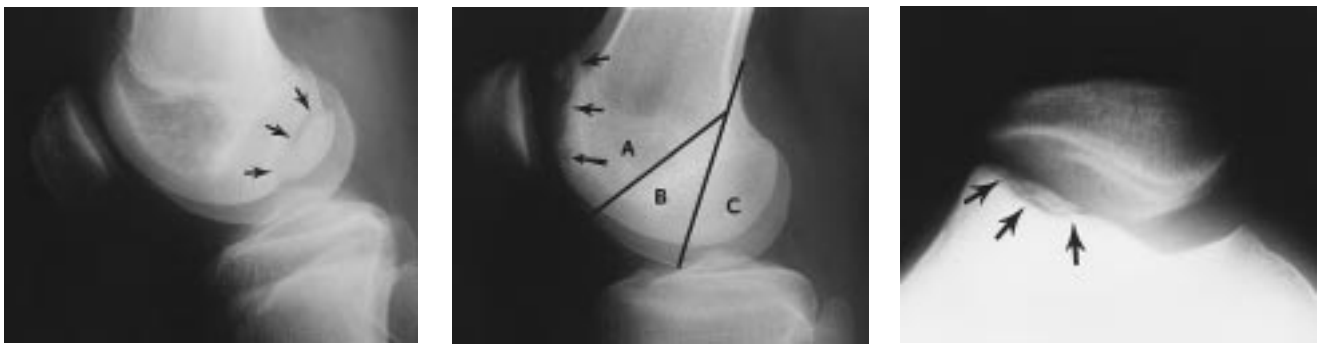

Fig. 7 Potentially inaccessible arthroscopic areas (arrows). **A,** Lateral radiograph of the knee of a 16-year-old male athlete who had been symptomatic for 22 months. The JOCD lesion located in area 1-2 C of the medial femoral condyle. A fragment mismatch and displacement can be seen. **B** and **C,** Images of the knee of a 15-year-old boy who had been symptomatic for 18 months. Lateral **(B)** and axial **(C)** radiographs show an arthroscopically inaccessible lesion in area 4-5 A of the condyle, with fragmentation of the lesion.

appearance of the lesion. In all cases, the lesion has a slight bronze tint, and the damage done to the articular cartilage by the metallic fixation is considerable (Fig. 8).

Although the functional significance of my observations with cannulated screws is uncertain, I have returned to the use of smooth pins. My technique is to bend the articular end of the pin to a right angle 1 mm from the end and to insert the pin through the lesion and the condyle so that it exits from the epicondylar region. The drill is then placed on the proximal end of the pin and is drilled out until only a few millimeters of the pin is visible on the articular or distal end. A clamp is then placed on the proximal end, and a small slap hammer is used to seat the bent distal end of the pin into the articular cartilage to the subchondral bone. This gives better fixation than can be achieved with the straight pins previously used. There has been no difficulty in removing these pins in a retrograde fashion through a small skin incision.

Fig. 8 The knee of a 19-year-old male patient who had a 6-year history of intermittent swelling and pain while participating in sports. At initial surgery, the anterior and lateral fragments were seen to be hinged, the posterior fragment was loose in the joint, and there were marked mismatches of all fragments. This intraoperative photograph was taken at the time of fixation removal. All three fragments had united, but discoloration of both femoral condyles and the screw cavities was seen. The posterior fragment had subsided in spite of a subchondral bone graft, perhaps due to screw compression. The long-term prognosis is poor.

Allografting

In the rare instances in which there is no alternative to removal of a JOCD lesion from a weight-bearing area, an allograft should be considered. Reasonable 5-year graft survival rates have been achieved with the use of both fresh and fresh-frozen osteochondral allografts.[17,18] Although there are risks of graft rejection and infection attendant to these procedures, the reality of early compartment degeneration of the knee following fragment removal should prompt consideration of an allograft.

Autologous Chondrocyte Implantation

The recent Swedish report on chondrocyte implantation[19] demonstrated good short-term results in articular cartilage defects of the femoral condyles. The development of this technology in the United States will soon make this biologic product available for the treatment of such defects.

Results

Many articles extolling the results of surgical treatment of JOCD/OCD have been published, all of them with less than 10 years of follow-up. The ultimate results of surgery for JOCD require long-term surveillance before it can be determined whether the treatment has been fully successful in preventing early-onset osteoarthritis. However, the poor results after removal of weight-bearing fragments in JOCD are apparent in follow-up periods as short as 8 years.

Summary

Both JOCD and OCD are frequently encountered by orthopaedic surgeons, especially those who have sports medicine practices. The pathologic changes of JOCD begin in subchondral bone, not the articular cartilage. Juvenile osteochondritis of the knee is the product of accumulated stresses to the femoral condyles resulting in subchondral stress fractures. Conservative management by activity restriction will succeed in about 50% of cases. Since most lesions occupy weight-bearing areas, their removal is not recommended. To avoid the complications of gonarthrosis, the objective of both conservative and surgical treatment must be to restore the joint surface to a normal configuration.

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References

1. König F: Ueber freie Körper in den Gelenken. *Dtsch Z Chir* 1887;27:90-109.
2. Barrie HJ: Osteochondritis dissecans 1887–1987: A centennial look at König's memorable phrase. *J Bone Joint Surg Br* 1987;69:693-695.
3. Lindén B: The incidence of osteochondritis dissecans in the condyles of the femur. *Acta Orthop Scand* 1976;47:664-667.
4. Aichroth P: Osteochondritis dissecans of the knee: A clinical survey. *J Bone Joint Surg Br* 1971;53:440-447.
5. Smillie IS: Treatment of osteochondritis dissecans. *J Bone Joint Surg Br* 1957;39: 248-260.
6. Green WT, Banks HH: Osteochondritis dissecans in children. *J Bone Joint Surg Am* 1953;35:26-47.

7. 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;17:601-606.
8. Lavner G: Osteochondritis dissecans: An analysis of forty-two cases and a review of the literature. *AJR Am J Roentgenol* 1947;57:56-70.
9. Aegerter E, Kirkpatrick JA Jr (eds): *Orthopedic Diseases: Physiology, Pathology, Radiology*, 3rd ed. Philadelphia: WB Saunders, 1968, pp 320-368.
10. Clanton TO, DeLee JC: Osteochondritis dissecans: History, pathophysiology and current treatment concepts. *Clin Orthop* 1982;167:50-64.
11. Cahill BR, Berg BC: 99m-Technetium phosphate compound joint scintigraphy in the management of juvenile osteochondritis dissecans of the femoral condyles. *Am J Sports Med* 1983;11:329-335.
12. Hughston JC, Hergenroeder PT, Courtenay BG: Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am* 1984;66:1340-1348.
13. Litchman HM, McCullough RW, Gandsman EJ, et al: Computerized blood flow analysis for decision making in the treatment of osteochondritis dissecans. *J Pediatr Orthop* 1988;8:208-212.
14. Miller RH III: Osteochondritis dissecans of femoral condyles, in Crenshaw AH (ed): *Campbell's Operative Orthopaedics*, 8th ed. St Louis: Mosby Year Book, 1992, vol 3, pp 1830-1835.
15. Sisk TD: Osteochondritis dissecans, in Crenshaw AH (ed): *Campbell's Operative Orthopaedics*, 8th ed. St Louis: Mosby Year Book, 1992, pp 1700-1707.
16. Convery FR, Meyers MH, Akeson WH: Fresh osteochondral allografting of the femoral condyle. *Clin Orthop* 1991; 273:139-145.
17. Mahomed MN, Beaver RJ, Gross AE: The long-term success of fresh, small fragment osteochondral allografts used for intraarticular posttraumatic defects in the knee joint. *Orthopedics* 1992;15:1191-1199.
18. Tomford WW, Springfield DS, Mankin HJ: Fresh and frozen articular cartilage allografts. *Orthopedics* 1992;15:1183-1188.
19. Brittberg M, Lindahl A, Nilsson A, et al: Treatment of deep cartilage defects in the knee with autologous chrondrocyte transplantation. *N Engl J Med* 1994;331; 889-895.