Recognition and Treatment of Osteochondritis Dissecans of the Femoral Condyles

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Abstract: Osteochondritis dissecans (OD) of the femoral condyles is a vague and often confusing diagnostic entity encountered by many clinicians. Unfortunately, there are several factors that add to this confusion. Chief among these is the proper recognition and understanding of the disease process, which is not well-documented. In addition, OD is often generically grouped together with other femoral condylar lesions that require differing diagnostic and treatment methods for proper care. OD is commonly divided into two categories, juvenile and adult forms. Each requires different methods of correction and rehabilitation. This paper describes the disease process of OD, explains the differences between the juvenile and adult forms (including common symptoms and diagnostic techniques), describes several of the pathologies that OD is mistakenly grouped with, and gives a brief review of the common arthroscopic and surgical techniques used to treat this pathology. In addition, rehabilitation guidelines and suggestions are offered to aid the athlete’s return to functional activities.

With the recent popularity of research dedicated to the knee, great strides have been made in understanding the many pathologies associated with the joint. One area, however, that is still controversial and confusing to many clinicians is osteochondritis dissecans (OD) of the femoral condyles. This disease process, first described by Paget, and named by Koenig, is often mistakenly grouped with several other distinctively different pathologies, which serve to add to the confusion surrounding it. OD can occur at many sites in the body, including the talus, mandible, elbow, shoulder, patella, and the femur.

The purposes of this paper are to describe the disease process that occurs with OD, differentiate its juvenile and adult forms, contrast OD to similar pathologies at the femoral condyles, discuss some commonly used arthroscopic and surgical interventions for OD, and to provide some suggestions for rehabilitating athletes suffering from OD.

The Disease Process

OD is a disease process of a mysterious, and usually controversial, nature. Several etiologies have been proposed including heredity and familial disposition, acute or repeated trauma, epiphyseal ossification abnormalities, avascular necrosis or impaired blood supply, and endocrine imbalances. Typically, OD is divided into two groups: juvenile, which is also commonly referred to as “early” or “under fifteen”, and adult, which is also called “late” or “over fifteen”. For purposes of simplification, these groups will be referred to as juvenile and adult throughout the body of this paper.

Juvenile OD is described as a single area of ossification developed separately from the main body of an otherwise normal epiphysis. Initial radiographic examination reveals an irregularity in the ossifying margin of the epiphysis. Later radiographs reveal a concentric flake of bone which grows at the same rate as the epiphysis yet remains separated from it by a transradiant line. The bone flake is usually several millimeters thick but has few, if any, trabeculae and has the general appearance of a non-union fracture.

Adult OD is described as a bone fragment or loose body that is usually a result of previous juvenile OD, although some insist later trauma can cause occurrence of the disease. The loose body results in a concave crater on the femoral condyle with steeply sloping edges.

Aichroth described five basic sites of OD lesions on the femoral condyles, with 85% of the lesions presenting on the medial condyle and 15% on the lateral condyle. Of these lesions, 69% occur on the “classic site,” which is the lateral aspect of the medial femoral condyle. Bradley and Dandy arthroscopically examined 5000 patients with varying lesions at multiple sites on the femoral condyles. The authors found what they described as true OD in both the juvenile and adult stages at only the classical site, despite a careful search for the condition at other areas. They suggested that the imprecise use of terminology regarding OD leads to much of the controversy surrounding it and that many of the patients generically termed as suffering from OD may actually be suffering from other nondissecting pathologies.

Symptoms and Diagnosis

As stated above, there are two distinct forms of OD, juvenile and adult, that can occur at various sites on the femoral condyles. Each of these presentations offer differing symptoms and treatment forms. In both the juvenile and adult forms, medial condylar lesions tend to be somewhat more anterior on the condyle and lead to patellofemoral joint pain and dysfunction of the extensor mechanism. Lateral condylar lesions

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often occur more posterior and lead to tibiofemoral derangement and are more prone to fragmentation.9

Juvenile OD may result in symptoms of nonspecific knee pain, mild effusion, quadiceps muscle atrophy, point tenderness, tibial external rotation with gait, and may have episodes of catching or locking.2,4,9,13 Bradley and Dandy4 point out that all of their juvenile OD cases occurred in the second decade of life and that a hemarthrosis occurred only in those patients with acute osteochondral fractures. Patients presenting with juvenile OD typically do not have a history of knee trauma.4,13 Diagnosis is usually made with a radiograph,9,13 but arthroscopy is considered an invaluable tool for assessing the progression of OD.9

Adult OD patients present with many of the same symptoms as the juvenile form but are more prone to catching or locking of the knee joint, "giving way" of the knee, and they may have a specific history of trauma.13 Bradley and Dandy4 pointed out that they did not see adult OD in any patients under the age of 18. Adult lesions tend to slough loose bodies creating a condylar defect which may be palpable with the knee flexed to 90°.13 Wilson's test is often positive for these patients as well.13,16 This test is performed with the patient sitting with the knees flexed over the edge of the examining table. The tibia is internally rotated and the knee actively extended. Pain increases at approximately 30° of knee flexion if there is an osteochondritis dissecans lesion at the classical site, and the patient is asked to stop the motion. The tibia is then externally rotated, and, if the pain disappears, the test is considered positive for OD.16 Radiographs, arthroscopy, three-dimensional CT scans, and MRI are used to confirm the diagnosis and to monitor its progress.4,7,9,13

Differentiating OD From Other Pathologies
OD can be differentiated from other sources of joint line pain during the physical examination. Meniscal and collateral ligament injuries can be assessed with testing in a routine physical examination.2,13 However, there are some specific condylar lesions that need to be defined and differentiated arthroscopically. Recently,4 many disorders that result in condylar fragment separation have been incorrectly termed OD; therefore, some clarification is warranted.

Idiopathic osteonecrosis is distinguished as a cavity created by the disappearance of subchondral bone roofed by a plate of cortical bone.4 The cortical bone roof is unstable and reveals a cavity containing shapeless soft tissue.

Chondral separations are full separations of articular cartilage that expose subchondral bone. Typically, chondral separations have very vertical margins.4 Very similar to chondral separations are chondral flaps, where there is partial separation of the articular cartilage but the subchondral bone is not exposed.

Osteochondral fractures are recognizable by having both a flat and a convex surface.4,9 Acute injuries cause a hemarthrosis, and the fragment is made of cancellous bone on the flat surface and a layer of articular cartilage of the convex surface. Old injuries have a loose body in the joint and a flattened region at the site of its origin.

In addition to these pathologies, steroid osteonecrosis and epiphyseal dysplasia can also cause fragmentation of the femoral condyles,4 but these conditions are usually quite distinguishable from the others.

Treatment of OD
Once OD is accurately recognized and diagnosed, it must be effectively treated. The standard treatment for juvenile OD is a period of rest lasting from 3 months to 1 year, and this is often enforced with immobilization or casting. Larger lesions may take on the appearance of adult OD and, as such, may require arthroscopy as the mode of treatment.9

Adult OD is usually treated arthroscopically. For a thorough coverage of the many procedures used arthroscopically, see Garrett.9

Simple drilling has been advocated as an effective mode of treatment but has recently been deemed controversial because the osteochondral fragment is usually removed within 1 to 2 years anyway.2,7,9 Retrograde drilling and bone grafting are suggested as a treatment method but are considered much more demanding and, at times, technically impossible and, as such, would not be suitable for the inexperienced surgeon.9

Extraction of the loose body is often performed and best indicated when the bone has fragmented, been altered in shape, or lacks sufficient bony backing to warrant other forms of stabilization.9,13

Replacement pinning of the loose body has long been the gold standard of treatment.9 However, this method is questionable.9 For the procedure to be fully effective, some bone grafting is required, and, in many cases, all one has left after the procedure is the equivalent of a stable, pinned, nonunion fracture that will not heal.9

Fixation has been performed with such items as smooth Kirschner wires, bone pegs, nails, biodegradable pins, and cannulated screws,2,9,13 but each of these items has some failing that makes it less than ideal. One fixation device that does seem to work well is the Herbert screw, which both compresses well and may be countersunk to allow early motion of the knee joint.9 The screws are relatively stiff, though, and must be removed early to prevent bone formation along the shank of the screw.

Abrasion arthroplasty and heterotopic autogenous grafting have also been used with good results on smaller lesions, but with little success on larger lesions.

Rehabilitation and Prognosis for OD Patients
Since the treatment for juvenile OD involves relatively lengthy immobilization and possible casting, many steps must be taken to counter the negative effects of this process. During this period, the young athlete must be encouraged to perform cardiovascular exercises such as the Upper Body Ergometer, seated Versaclimber exercise, or other sim-
ilar aerobic activities. Lower extremity flexibility should be maintained, and every major muscle group, with the exception of the quadriceps, can be safely stretched while maintaining immobilization of the knee joint.

Strength can be maintained through use of such exercises as “four-way” straight leg raises (hip flexion/adduction/abduction/extension) and ankle tubing exercises. Quadriceps and hamstring coactivation, or setting, can be performed while in an immobilizer or cast. The use of neuromuscular electrical stimulation to the quadriceps and hamstring strings for coactivation contractions can further augment the strength maintenance program.15

Following immobilization, the techniques described above should be continued, and range of motion exercises and joint mobilizations of the knee, as well as progressive quadriceps and hamstring strengthening should be performed. Weight-bearing progression throughout rehabilitation should be to patient tolerance, and aquatic therapy is very beneficial in facilitating the return to full-weight-bearing status. Gait training techniques, such as manual facilitation and visual feedback to the patient via a full-length mirror, may be used to address any gait deviations that developed during the immobilization and decreased weight-bearing phases of rehabilitation. In addition, exercises to restore normal knee and ankle joint proprioception, such as biomechanical ankle platform systems (BAPS board) exercises or unilateral stance, are also beneficial to the athlete planning to return to competition.

The prognosis for juvenile OD is very good as most conditions result in excellent healing and a complete restoration of normal joint mechanics.4,9

The rehabilitation for adult OD differs in that most of these cases are surgical. The surgical procedure will have an impact on the rehabilitation guidelines but, generally, continuous passive motion is used very early after surgery, and, for minor lesions, immediate weight bearing is possible. Larger lesions (over 3 cm) may demand weight-bearing restrictions for up to 6 weeks.9 Fortunately, the physical therapist or athletic trainer does not have the immobilization concerns that occur in the juvenile form. However, the overall prognosis is not as good in the adult OD patient.4,13 Crawfurd et al7 reported that only 30% of patients had successful spontaneous healing of stable lesions at the classic site when followed up 7.5 years after the initial arthroscopic diagnosis. Athletic activity may be restricted for up to 6 months and some repetitive trauma sports such as basketball and distance running may be discouraged by the physician due to the likelihood of reinjury. Regardless, a thorough rehabilitation program, such as that discussed for juvenile OD, should be developed and implemented to assist in the potential return to competitive athletics.

Acknowledgments

I would like to thank Joe Gieck, EdD, PT, ATC, and Doug Keskula, PhD, PT, ATC, for their assistance in reviewing and editing this manuscript for publication.

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