

Osteochondritis dissecans of the knee

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Abstract

Osteochondritis dissecans (OCD) of the knee is a common cause of knee pain and dysfunction among skeletally immature and young adult patients. OCD is increasingly frequently seen in pediatric, adolescent and young adult athletes. If it is not recognized and treated appropriately, it can lead to secondary osteoarthritis with pain and functional limitation. Stable lesions in skeletally immature patients should initially be managed non-operatively. Unstable juvenile lesions and stable juvenile lesions that fail to heal with non-operative treatment require a surgical treatment. By contrast, adult OCD of the knee rarely responds to conservative measures because of limited healing potential. Operative treatment depends on the lesion stage, and there exist several surgical options.

Key words: cartilage, knee, osteochondral, osteochondritis dissecans.

Introduction

Osteochondritis dissecans (OCD) is a common cause of knee pain and dysfunction among skeletally immature and young adult patients. OCD is an acquired idiopathic lesion of subchondral bone that may produce delamination and sequestration with or

without articular cartilage involvement (1-5). The etiology of OCD remains controversial. It is most widely considered to be due to repeated microtraumas associated with vascular impairment, as supported by the fact that the lesion is classically located on the lateral aspect of the medial femoral condyle, where it may be due to contact with a hypertrophic tibial spine (6).

Other possible causes are defects of ossification, repeated mechanical stress and ischemia (7, 8). The lesion may heal spontaneously or a fragment may detach and become displaced in the joint cavity, resulting in the presence of an intra-articular loose body (8, 9).

If OCD is not recognized and treated appropriately, it may lead to secondary osteoarthritis (OA) with pain and functional limitation. Radiography shows that patients with adult OCD develop knee OA about 10 years earlier than primary osteoarthritic patients (10).

Classification

OCD is usually classified as “juvenile” or “adult” depending on the maturity of the distal femoral physis. Most cases of adult OCD are thought to be due to persistence of an unresolved juvenile OCD lesion, although *de novo* adult OCD lesions have also been described (1).

Adult OCD lesions have a propensity for instability and typically follow a progressive and unremitting clinical course. By comparison, juvenile OCD lesions with an intact articular surface have the potential to heal (11-13). Both adult and juvenile OCD lesions that do not heal can potentially be associated with sequelae later on, such as premature degenerative joint disease (10).

Lesions assessed arthroscopically can be classified, according to their severity, using the International Cartilage

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Repair Society (ICRS) scale of OCD lesions (14):

Type I: Stable lesion with a continuous but softened area covered by intact articular cartilage

Type II: Lesion with partial articular cartilage discontinuity, stable when probed

Type III: Lesion with complete articular cartilage discontinuity, but no dislocation (“dead *in situ*”)

Type IV: Empty defect, or defect with a dislocated fragment or loose fragment within the bed (Fig. 1).

The prognosis as regards the healing of an OCD lesion depends on both skeletal maturity and the severity of the lesion. In younger patients with an open physis, the prognosis is more favorable.

Epidemiology

Even though the exact prevalence of OCD is unknown, rates of between 15 and 29 per 100,000 have been reported (10, 15).

The condition shows a male preponderance (a male:female ratio of 5:3), although its prevalence among girls is increasing. The mean age at OCD onset may be decreasing since the incidence of the condition appears to be influenced by the growing participation of children in competitive sports (1).

More than 70% of OCD lesions are found in the posterolateral aspect of the medial femoral condyle,

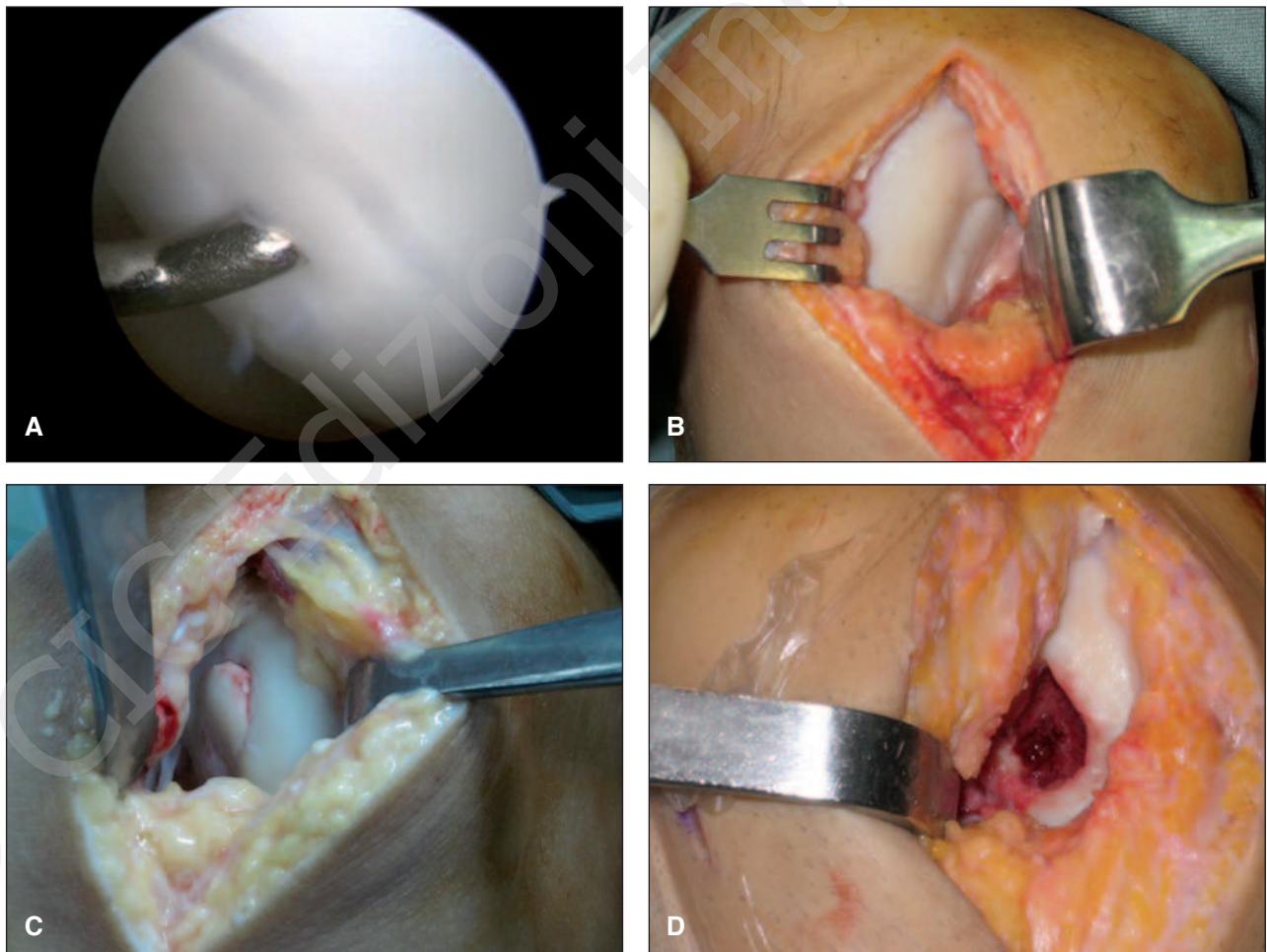


Fig. 1. ICRS classification of OCD lesions. A: Type I: stable lesion with a continuous but softened area covered by intact articular cartilage. B: Type II: lesion with partial articular cartilage discontinuity, stable when probed. C: Type III: lesion with complete articular cartilage discontinuity, but no dislocation (“dead *in situ*”). D: Type IV: empty defect, or defect with a dislocated fragment or loose fragment within the bed.

15% in the inferior central aspect of the lateral condyle and less than 1% in the trochlea. Patellar lesions, typically located in the inferior medial area, are uncommon (5-10%).

Etiology

The possible causes of OCD (traumatic, ischemic, idiopathic and hereditary) are still debated, however most authors now believe that OCD has a multifactorial etiology (16).

Trauma

Trauma is the most recognized cause of OCD. Although a direct trauma to the knee could result in a transchondral fracture, the classic location of OCD in the posterolateral portion of the medial femoral condyle suggests that indirect trauma is a more likely cause. Repeated impingement of the tibial spine on the lateral aspect of the medial femoral condyle during internal rotation of the tibia has also been suggested to be a contributing factor (6).

Ischemia

Ischemia has been investigated as a potential cause of OCD. Enneking (17) found the vascular supply to the subchondral bone to be similar to the vascular supply to the bowel mesentery, with poor anastomoses to surrounding arterioles. This propensity for ischemia would naturally lead subchondral bone to form sequestra, making it particularly vulnerable to traumatic insult, resultant fracture, and potential separation. Instead, Rogers and Gladstone (18) studied the vascularity of the distal part of the femur and found numerous anastomoses to intramedullary cancellous bone.

Genetics

Several authors have investigated a potential genetic link for OCD. Petrie (16) found no clear genetic etiology for OCD.

Clinical presentation

Knee OCD lesions, early in their course, are associated with vague and poorly defined symptoms, including

variable amounts of pain and swelling. As the lesion progresses, symptoms such as catching, locking and giving way appear and increase in frequency. Symptoms that are constant and severe are typically associated with loose bodies within the knee. Affected patients may note locking and may be able to palpate the loose body in the affected joint.

Imaging

Imaging studies are crucial for characterizing OCD lesions, assessing lesion status and predicting prognosis. An ideal imaging algorithm would assist the surgeon in distinguishing surgical from non-surgical cases.

Radiographic evaluation is an appropriate first-line investigation. As described by Cahill and Berg (19), lesion location and size can be established from a radiograph. Initial imaging must include anteroposterior and lateral views of the knee. A notch or tunnel posteroanterior radiograph is the best way to visualize a lesion in the medial femoral condyle. A skyline view should be added when patellar involvement is suspected.

Magnetic resonance imaging (MRI) is a routine part of the diagnostic evaluation of OCD. It can be used to visualize loose bodies and to determine the degree of displacement. MRI also allows evaluation of the fluid interfaces and of the integrity of the articular surfaces. MRI can be used to accurately estimate the lesion size and to evaluate the status of cartilage and subchondral bone; OCD lesions can also be identified on the basis of the presence of a high signal zone beneath the lesion, the extension of bony edema (Fig. 2), and the presence of loose bodies, which are characteristic of these lesions (13).

De Smet et al. (20) described four criteria, on T2-weighted MRI studies, that correlated with the ability of OCD lesions to heal after non-operative treatment: i) a line of high signal intensity at least 5 mm in length between the OCD lesion and underlying bone, ii) an area of increased homogeneous signal, at least 5 mm in diameter, beneath the lesion, iii) a focal defect of 5 mm or more in the articular surface, and iv) a high signal line traversing the subchondral plate into the lesion. Of these signs, the high signal line behind the fragment, found in 72% of unstable lesions, emerged

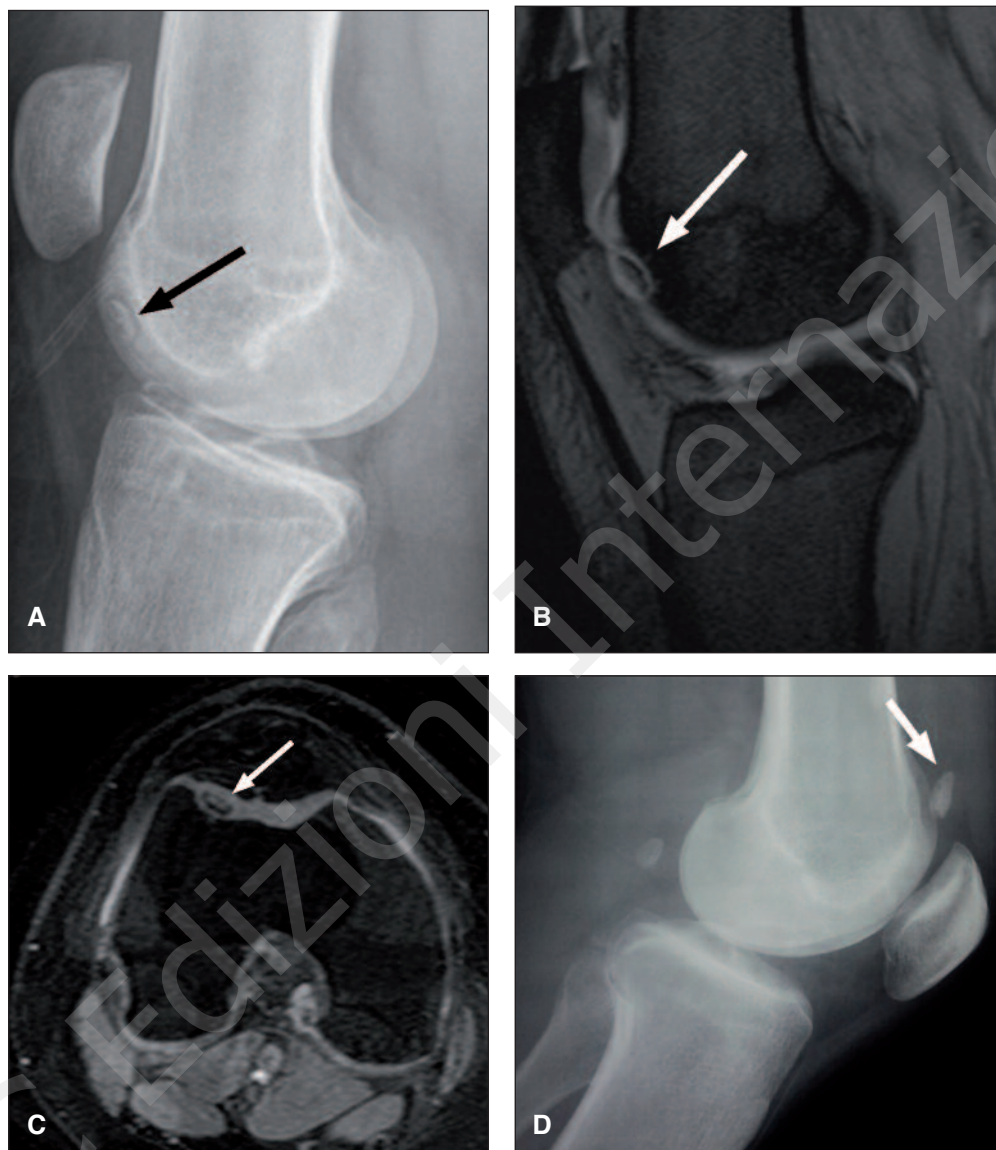


Fig. 2. Lateral radiograph (A), sagittal MRI (B) and axial MRI (C) of osteochondritis dissecans lesion of the trochlea (arrow). The lesion is clearly demarcated from underlying subcondral bone with apparent anterior separation of articular surface. D: Lateral radiograph showing a loose body (arrow).

as the most predictive (20). Pill et al. (21) found the high signal line to be the most common sign in patients who failed non-operative treatment.

Intra-articular injection of gadolinium increases the sensitivity and specificity of MRI findings (22). Bohndorf (23) demonstrated that enhancement of the high signal line behind the fragment after intravenous gadolinium administration indicated healing granulation tissue, not synovial fluid. Technetium bone scintigraphy is an important tool for evaluating the potential healing of the osteochondral fragment. Increased uptake is correlated with osteoblastic activity. Technetium imaging may also reveal occult bilateral involvement. Paletta et al. (24) reviewed quantitative bone scans in a small series (12 patients) and found that increased activity predicted healing in 100% of patients with open femoral physes, but not in adolescents with closing physes. Computed tomography

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(CT) scanning may be helpful in preoperative planning and in guiding treatment when MRI is not available or is contraindicated.

Conservative treatment

Juvenile OCD with stable lesions frequently responds to non-operative treatment, such as immobilization, non-weight-bearing or limitation of activity (25).

Cahill (1) reported that 50% of juvenile OCD lesions will heal within a 10- to 18-month period, provided the physis remains open and patient compliance is maintained. Symptomatic lesions in children, or skeletally immature patients, should initially be treated with conservative measures for three months, if there are no loose bodies upon radiographic examination. However, surgical treatment is indicated if non-operative treatment has failed over a sustained period of time (usually six months for the juvenile type or a shorter period for the adult type) or if there is an unstable lesion. By contrast, adult OCD of the knee rarely responds to conservative measures.

Surgical treatment

Operative treatment should be considered in skeletally immature patients with detached or unstable lesions and in those patients approaching physal closure whose lesions have been unresponsive to non-operative management (1, 26, 27). By contrast, the majority of adult OCD lesions are unstable and the clinical course more deleterious, necessitating early, aggressive surgical intervention (28, 29).

Patients with lesions <2 cm in diameter should be offered arthroscopic intervention in the form of subchondral drilling and, possibly, debridement and fragment stabilization. Bone grafting may be required.

Patients with lesions >2 cm in diameter or patients with multiple loose bodies (Fig. 3) may have their lesions fixed through an open procedure. Patients may also be offered autologous chondrocyte implantation or mosaicplasty.

Patients with lesions having a diameter of 2 cm or more may be offered radical removal of sclerotic bone

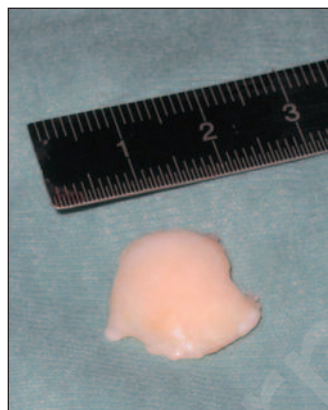


Fig. 3. Excision of a chronic loose body.

with bone grafting of the defect and autologous chondrocyte implantation (sandwich technique).

Arthroscopic subchondral drilling

With this method, multiple perforations of the lesion are made using Kirschner wires. While arthroscopic drilling of juvenile OCD lesions is well docu-

mented in the literature, its application in adult OCD is less well recognized. The two most common techniques are transarticular drilling and retroarticular drilling, both of which serve to create channels for potential revascularization and healing (30, 31). Although transarticular drilling is accurate and technically easy, it creates articular cartilage channels that heal with fibrocartilage. With retroarticular drilling, on the other hand, such articular cartilage damage can be avoided. However, this technically demanding procedure can result in inadequate drilling into the defect and soft-tissue injury and it has usually been performed in conjunction with bone grafting (28, 32). To perform bone grafting, additional invasive surgery is required in order to harvest autogenous cancellous bone, usually from the iliac crest. A number of authors have found arthroscopic transarticular drilling to be effective in treatment of OCD lesions in skeletally immature patients but less effective in those in whom physal closure has already occurred (30, 33-36).

Arthroscopic debridement and fragment stabilization

The literature contains descriptions of a number of surgical techniques for the operative fixation of unstable OCD lesions in the knee using bioabsorbable nails or pins and non-absorbable screw fixation (37-39).

In cases with unstable lesions, fibrous tissue found between the fragments should be removed. If bone loss is significant, cancellous bone grafting may be required. If partially unstable lesions have subchondral bone loss, autogenous bone graft is packed into the crater before fragment reduction and fixation. Herbert screws and

cannulated screws have been used successfully, with second surgeries required for removal (40, 41).

Although several authors have reported positive results with bioabsorbable screws, this type of surgical intervention is not devoid of complications, related to biodegradation of the synthetic polymers and host response (42-44).

Transplantation of autologous osteochondral plugs for defect replacement in skeletally mature patients is well documented in the literature. However, this procedure has potential drawbacks, namely donor site morbidity and incongruent articular fit (45-47).

Autologous osteochondral mosaicplasty

This technique involves the transplantation of cylindrical osteochondral grafts, taken from non-weight-bearing regions of the knee, in order to reconstruct a weight-bearing surface or affected area. Combinations of different graft sizes are used to allow a greater filling rate.

Autologous osteochondral mosaicplasty was found to be 94% effective for OCD lesions requiring surgery in the femoral condyle. However, a prospective, randomized study directly compared autologous chondrocyte implantation with mosaicplasty for OCD lesions of the knee and found that autologous chondrocyte implantation gave significantly better results (45).

Osteochondral allograft transplantation involves the use of a freshly harvested allograft condyle. The advantages are that the exact condyle curvature can be reconstructed and no further defect is created during harvesting.

Autologous chondrocyte implantation

Autologous chondrocyte implantation involves a diagnostic arthroscopy, the harvesting of a small amount of cartilage cells for cell culture, and subsequent reimplantation of cells into the defect. Newer surgical treatments, including single-stage cell-based procedures, use mesenchymal stem cells and matrix augmentation (48). Autologous chondrocyte implantation has been used to treat large, isolated femoral defects in skeletally mature patients. If there is a lack of subchondral bone in addition to articular cartilage, bone grafting of the OCD crater is often necessary prior to implantation.

Peterson et al. (49) reported successful clinical results in more than 90% of patients. Bentley et al. (45)

reported significantly superior outcomes with autologous chondrocyte implantation over autologous plugs for osteochondral defects in adult knees; moreover, the mosaicplasty plug results deteriorated over time.

Radical removal of sclerotic bone with bone grafting of the defect and autologous chondrocyte implantation (sandwich technique)

The defect is excised to the normal surrounding cartilage, and the sclerotic bone is excavated down to the bleeding cancellous bone. The osseous defect is filled with cancellous bone to the subchondral bone plate, and a periosteal flap is harvested and used to secure the graft. A second periosteal flap is then used for the autologous chondrocyte implantation, which is performed as previously described.

New surgical treatments

New surgical treatments, including a single-stage procedure, use a biomimetic osteochondral scaffold (a three-layer collagen-hydroxyapatite scaffold) (Fig. 4). Filardo et al. (50) treated 27 patients with symptomatic knee OCD of the femoral condyles with the implantation of a three-layer collagen-hydroxyapatite scaffold. A statistically significant improvement in all clinical scores was obtained at one-year and two-year follow-up.



Fig. 4. Surgical view: osteochondral scaffold implantation at the medial femoral condyle.

References

- Cahill BR. Osteochondritis dissecans of the knee: treatment of juvenile and adult forms. *J Am Acad Orthop Surg*. 1995;3:237-247.
- Clanton TO, DeLee JC. Osteochondritis dissecans. History, pathophysiology and current treatment concepts. *Clin Orthop Relat Res*. 1982; (167):50-64.
- Glancy GL. Juvenile osteochondritis dissecans. *Am J Knee Surg*. 1999;12:120-124.
- Kocher MS, Micheli LJ. The pediatric knee: evaluation and treatment. In: Insall JN, Scott WN, eds. *Surgery of the Knee*. 3rd ed. New York, NY: Churchill-Livingstone; 2001:1356-1397.
- Pappas A. Osteochondrosis dissecans. *Clin Orthop Relat Res*. 1981; (158):59-69.
- Pape D, Filardo G, Kon E, et al. Disease-specific clinical problems associated with the subcondral bone. *Knee Surg Sports Traumatol Arthrosc*. 2010;18:448-462.
- Obedian RS, Grelsamer RP. Osteochondritis dissecans of the distal femur and patella. *Clin Sports Med*. 1997;16:157-174.
- Schenck RC Jr, Goodnight JM. Osteochondritis dissecans. *J Bone Joint Surg Am*. 1996; 78:439-456.
- O'Driscoll SW. The healing and regeneration of articular cartilage. *J Bone Joint Surg Am*. 1998;80:1795-1812.
- Lindén B. The incidence of osteochondritis dissecans in the condyles of the femur. *Acta Orthop Scand*. 1976;47:664-667.
- 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.
- Green WT, Banks HH. Osteochondritis dissecans in children. *J Bone Joint Surg Am* 1953; 35-A:26-47.
- Hefli F, Beguiristain J, Krauspe R, et al. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. *J Pediatr Orthop B*. 1999; 8:231-245.
- Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am*. 2003;85 (Suppl 2):58-69.
- Hughston JC, Hergenroeder PT, Courtenay BG. Osteochondritis dissecans of the femoral condyles. *J Bone Joint Surg Am*. 1984;66:1340-1348.
- Petrie PW. Aetiology of osteochondritis dissecans. Failure to establish a familial background. *J Bone Joint Surg Br*. 1977; 59:366-367.
- Enneking WF. *Clinical Musculoskeletal Pathology*. Ed. 3. Gainesville, Florida: University of Florida Press, 1990:166.
- Rogers WM, Gladstone H. Vascular foramina and arterial supply of the distal end of the femur. *J Bone Joint Surg Am*. 1950;32 (A:4):867-874.
- Cahill BR, Berg BC. 99m-Tc-phosphate compound joint scintigraphy in the management of juvenile osteochondritis dissecans of the femoral condyles. *Am J Sports Med*. 1983;11:329-335.
- De Smet AA, Ilahi OA, Graf BK. Untreated osteochondritis dissecans of the femoral condyles: prediction of patient outcome using radiographic and MR findings. *Skeletal Radiol*. 1997;26:463-467.
- Pill SG, Ganley TJ, Milam RA, et al. Role of magnetic resonance imaging and clinical criteria in predicting successful nonoperative treatment of osteochondritis dissecans in children. *J Pediatr Orthop*. 2003;23:102-108.
- Choi YS, Cohen NA, Potter HG, et al. Magnetic resonance imaging in the evaluation of osteochondritis dissecans of the patella. *Skeletal Radiol*. 2007;36: 929-935.
- Bohndorf K. Osteochondritis (osteochondrosis) dissecans: a review and new MRI classification. *Eur Radiol*. 1998;8:103-112.
- Paletta GA Jr, Bednarz PA, Stanitski CL, et al. The prognostic value of quantitative bone scan in knee osteochondritis dissecans: a preliminary experience. *Am J Sports Med*. 1998;26:7-14.
- Twyman RS, Desai K, Aichroth P. Osteochondritis dissecans of the knee: a long-term study. *J Bone Joint Surg Br*. 1991;73:461-464.
- Ewing JW, Voto SJ. Arthroscopic surgical management of osteochondritis dissecans of the knee. *Arthroscopy*. 1988;4:37-40.
- Guhl JF. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res*. 1982; (167): 65-74.
- Garrett JC. Osteochondritis dissecans. *Clin Sports Med*. 1991;10:569-593.
- Michael JW, Wurth A, Eysel P, et al. Long-term results after operative treatment of osteochondritis dissecans of the knee joint-30 year results. *Int Orthop*. 2008;32:217-221.
- Bradley J, Dandy DJ. Results of drilling osteochondritis dissecans before skeletal maturity. *J Bone Joint Surg Br*. 1989;71:642-644.
- Kawasaki K, Uchio Y, Adachi N, et al. Drilling from the intercondylar area for treatment of osteochondritis dissecans of the knee joint. *Knee*. 2003;10:257-263.
- Lebolt JR, Wall EJ. Retroarticular drilling and bone grafting of juvenile osteochondritis dissecans of the knee. *Arthroscopy*. 2007; 23:794.e1-794.e4.
- Aglietti P, Buzzi R, Bassi PB, et al. Arthroscopic drilling in juvenile osteochondritis dissecans of the medial femoral condyle. *Arthroscopy*. 1994;10:286-291.
- Anderson AE. Antegrade drilling for osteochondritis dissecans of the knee. *Arthroscopy*. 1997;13:319-324.
- Ganley TJ, Amro RR, Gregg JR, et al. Antegrade drilling for osteochondritis dissecans of the knee. Paper presented at: Pediatric Orthopaedic Society of North America 2002 Annual Meeting; May 3-5, 2002; Salt Lake City, Utah.
- Kocher MS, Micheli LJ, Yaniv M, et al. Functional and radiographic outcome of juvenile osteochondritis dissecans of the knee treated with transarticular arthroscopic drilling. *Am J Sports Med*. 2001;29:562-566.
- Weckström M, Parviainen M, Kiuru MJ, et al. Comparison of bioabsorbable pins and nails in the fixation of adult osteochondritis dissecans fragments of the knee: an outcome of 30 knees. *Am J Sports Med*. 2007;35:1467-1476.
- Tabaddor RR, Banffy MB, Andersen JS, et al. Fixation of juvenile osteochondritis dissecans lesions of the knee using poly 96L/4D-lactide copolymer bioabsorbable implants. *J Pediatr Orthop*. 2010;30:14-20.
- Anderson AE, Lipscomb AB, Coulam C. Antegrade curettage, bone grafting, and pinning of osteochondritis dissecans in the skeletally mature knee. *Am J Sports Med*. 1990;18: 254-261.
- Cugat R, Garcia M, Cusco X, et al. Osteochondritis dissecans: a historical review and its treatment with cannulated screws. *Arthroscopy*. 1993; 9: 675-684.
- Johnson LL, Uirtlugt G, Austin MD, et al. Osteochondritis dissecans of the knee: arthroscopic compression screw fixation. *Arthroscopy*. 1990; 6:179-189.
- Dervin GF, Keene GC, Chissell HR. Biodegradable rods in adult osteochondritis dissecans of the knee. *Clin Orthop Relat Res*. 1998; (356):213-221.
- Friederichs MG, Greis PE, Burks RT. Pitfalls associated with fixation of osteochondritis dissecans fragments using bioabsorbable screws. *Arthroscopy*. 2001;17:542-545.

44. Kim SJ, Shin SJ. Loose bodies after arthroscopic osteochondral autograft in osteochondritis dissecans of the knee. *Arthroscopy*. 2000;16: E16.
45. Bentley G, Biant LC, Carrington RW, et al. A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br*. 2003;85:223-230.
46. Outerbridge HK, Outerbridge AR, Outerbridge RE. The use of a lateral patellar autologous graft for the repair of a large osteochondral defect in the knee. *J Bone Joint Surg Am*. 1995; 77:65-72.
47. Yoshizumi Y, Sugita T, Kawamata T, et al. Cylindrical osteochondral graft for osteochondritis dissecans of the knee: a report of three cases. *Am J Sports Med*. 2002;30:441-445.
48. Erickson BJ, Chalmers PN, Yanke AB, et al. Surgical management of osteochondritis dissecans of the knee. *Curr Rev Musculoskelet Med*. 2013;6:102-114.
49. Peterson L, Minas T, Brittberg M, et al. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation: results at two to ten years. *J Bone Joint Surg Am*. 2003;85-A Suppl 2:17-24.
50. Filardo G, Kon E, Di Martino A, et al. Treatment of knee osteochondritis dissecans with a cell-free biomimetic osteochondral scaffold: clinical and imaging evaluation at 2-year follow-up. *Am J Sports Med*. 2013;41:1786-1793.