Knee Cartilage Tibio-Femoral Injuries

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Summary: Articular cartilage lesions of the femoral condyles are common and have limited healing capacity. The goal of surgical management is to relieve pain and mechanical symptoms, and hopefully slow the progression of osteoarthritis. Numerous surgical procedures have been described to include chondroplasty, microfracture, autologous chondrocyte implantation, autologous osteochondral transfer, allograft osteochondral transplantation, and fragment fixation. Each technique is distinguished by number of procedures required, expense, donor-site morbidity, risks of allograft tissues, and rehabilitation requirements. To maximize the surgical outcomes, it is imperative to treat any concomitant pathology to include mechanical malalignment, ligamentous instability, and/or meniscal deficiency. Several treatment algorithms have been proposed to guide management of symptomatic, focal chondral injuries of the femoral condyles. Algorithm decisions are based on lesion location, size, patient activity level, and earlier treatment. This article reviews the treatment considerations of the various techniques and the published results and illustrative case examples.

Key Words: cartilage—chondral—osteochondral—knee—treatment.

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Articular cartilage injuries involving the knee are common, occurring in up to 60% of arthroscopic evaluations.[1] Tibiofemoral articulation involvement is common and most often identified on the medial femoral condyle.[2] The natural history of untreated osteochondral lesions is unpredictable with regard to disease progression and subsequent development of osteoarthritis. Symptomatic progression is likely dependent on multiple injury and patient characteristics; to include, chondral injury size, location, depth, concomitant intra-articular pathology, limb alignment, patient age, activity level, and comorbid conditions.

Articular cartilage has a limited healing capacity because of the relative avascularity and the subsequent separation from pluripotential cells. As a result, osteochondral defects can be filled with biomechanically inferior fibrocartilage composed primarily of type II collagen. The osteochondral defect or fibrocartilage response may cause pain, mechanical symptoms, and alter articular contact forces, which may lead to progressive articular cartilage degradation.[3] The goal of intervention is to reduce pain and mechanical symptoms, restore articular congruity, and possibly prevent further injury.

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A number of surgical options are available for management of articular cartilage injuries. These options cover a wide range in patient morbidity, cost, rehabilitation requirements, and technical difficulty. Although a variety of cartilage procedures have been described, the most commonly performed procedures can be grouped into arthroscopic debridement (chondroplasty), fragment fixation, marrow stimulation, osteochondral grafting (allograft and autograft), and autologous chondrocyte implantation (ACI).

GENERAL CONSIDERATIONS

Articular cartilage injuries are managed while considering injury and patient characteristics. Patient-specific variables to consider are patient age, symptoms, activity limitations, treatment history, future activity or sport demands, and ability to participate in rehabilitation. In addition, comorbidities, social history (tobacco use), and weight (body mass index) can significantly affect treatment options and clinical outcomes.

Indications for operative management of articular cartilage injuries are symptomatic, focal cartilage lesions in a physiologically young patient. Chronologic age in the fifth decade has been cited as a relative contraindication to a cartilage restoration procedure because of presumed diffuse disease, short-term relief of symptoms, and the success of arthroplasty solutions.[4] However, our decision algorithm is based more on physiologic age, activity level, and our assessment of the osteochondral lesion. Successful treatment of osteochondral lesions is predicated on first addressing associated pathologic conditions, such as ligamentous stability, meniscal deficiency, and limb malalignment.

EVALUATION

Preoperative evaluation is initiated with a detailed history and physical examination. Specific information to illicit is a history of trauma, details surrounding the onset of symptoms, presence of mechanical symptoms, and complaints of instability. The location, character, and severity of the chief complaint must be attributable to the identified chondral injury to ensure improvement with surgery. Physical examination of the knee for chondral injuries of the tibio-femoral articulation is focused on excluding other pathology. A thorough knee examination evaluating for effusion, crepitus, meniscal injury, or ligamentous stability will ensure that all pathology is addressed and increase the chance of successful clinical outcome.

Preoperative radiographic work-up includes a weight-bearing anteroposterior, 45-degrees flexion posteroanterior, lateral, and merchant views. We routinely obtain full-length standing bilateral hip to ankle alignment radiographs on any patients with chondral lesions. Magnetic resonance imaging (MRI) is useful for evaluating cartilage lesions and concomitant intra-articular pathology. T2 and Proton-density weighted fast spin echo and T1-weighted gradient echo sequences provide the best visualization of articular cartilage lesions.[5]

TABLE 1. ICRS and Modified Outerbridge Classification for Articular Cartilage Injury

	ICRS	Modified Outerbridge
Grade 0	Normal	
Grade 1	Soft indentation, superficial fissures, and cracks	Softening and swelling
Grade 2	Lesions extending down $< 50\%$ of depth	Fragmentation and fissuring; < 15 mm in diameter
Grade 3	A. $> 50\%$ of depth B. Down to calcified layer C. Down to subchondral bone D. Cartilage blistering	Fragmentation and fissuring to subchondral bone; > 15 mm in diameter
Grade 4	Through subchondral bone	Exposed subchondral bone

ICRS indicates International Cartilage Repair Society.

MRI can also be used to assess osteochondritis dissecan (OCD) lesions and assist with preoperative planning.[5–8]

Chondral injuries have been classified by multiple investigators as a means of improving communication between physicians, guide management, and provide a framework for academic discussion.[5,9–11] A modification of the outerbridge classification or the International Cartilage Repair Society is most commonly used (Table 1).

TREATMENT

Initial nonoperative management includes rest, analgesics, nonsteroidal antiinflammatory medications, activity restrictions, and physical therapy for strength and conditioning. MRI or clinical evidence of an intraarticular loose body or an unstable OCD lesion warrants early surgical intervention. There is no evidence to support intra-articular steroid injections, haluronic acid, or glucosamine and chondrotin for the treatment of focal cartilage lesions. For physiologically older patients with diffuse osteoarthritis or 1 compartment disease, total knee arthroplasty or unicompartmental knee arthroplasty should be considered.

A number of useful algorithms have been published for selection of the appropriate procedure to treat cartilage defects.[4,12–14] Numerous patient and injury factors must be considered to offer the appropriate intervention to a patient. The investigators prefer the use of the treatment algorithm proposed by Cole et al (Fig. 1).[4] Although this approach focuses on the size of the chondral defect and the activity level of the patient, it does not account for the myriad of factors that are typically considered when treating these patients. However, it does provide a foundation from which these lesions can be approached. A dichotomization is made based upon activity into "low-demand" and "high-demand" patients. Military patients (despite athletic activity or job classification) can all be considered high-demand patients.

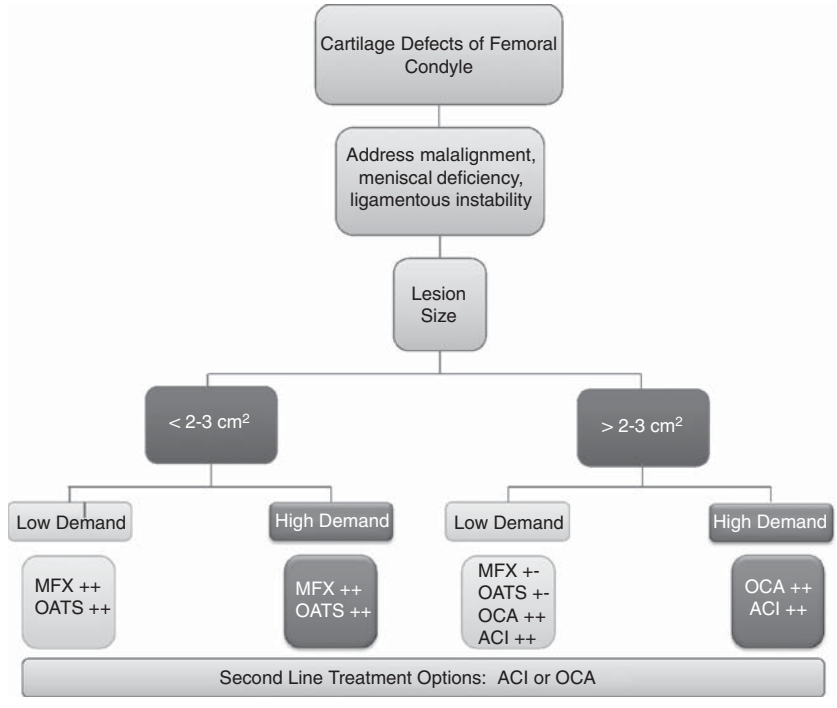

FIGURE 1. Treatment algorithm for chondral effects of the femoral condyle. Adapted with permission from *J Bone Joint Surg Am.* 2009;91:1778–1790. ACI indicates autologous chondrocyte implantation; OATS, osteoarticular transfer system; OCA, osteochondral allografting; MFX, microfracture.

Limb alignment, ligamentous instability, and meniscal pathology should be addressed when treating focal cartilage injuries. These procedures can be performed at the same surgical setting or before surgical management of the cartilage injury in a staged manner. If a staged procedure is planned, the osteotomy should preceed ligamentous or chondral work. Surgical outcomes from all of the cartilage treatment procedures have worse outcomes because of shear forces and increased contact stresses associated with these conditions. The investigators routinely perform hip to ankle alignment radiographs to assess malalignment and rely on physical examination and MRI to rule out meniscal and/or ligament deficiency. Medial compartment chondral disease in patients with varus malalignment should be treated in conjunction with an opening wedge high-tibial osteotomy (Figs. 2A–E). The patients with lateral compartment disease and valgus malalignment should undergo distal femoral osteotomy (Figs. 3A–G). Ligamentous instability should be corrected with cruciate and/or collateral reconstructions. Meniscal deficiency should be corrected with meniscal allograft transplantation (Figs. 3A–G). Once the mechanical alignment and concomitant meniscal and ligamentous deficiency is corrected, the appropriate chondral technique is selected.

Chondroplasty

Arthroscopic debridement and lavage is the first-line arthroscopic treatment of cartilage injuries. It does not require additional instrumentation, has a low morbidity to the patient, and allows for a rapid return to activity. The goal of the procedure is to remove loose flaps of cartilage, smooth irregular cartilaginous borders, and to lavage inflammatory cytokines from the joint.[15] The procedure is performed with a standard arthroscopic shaver. Loose bodies, unstable flaps, and

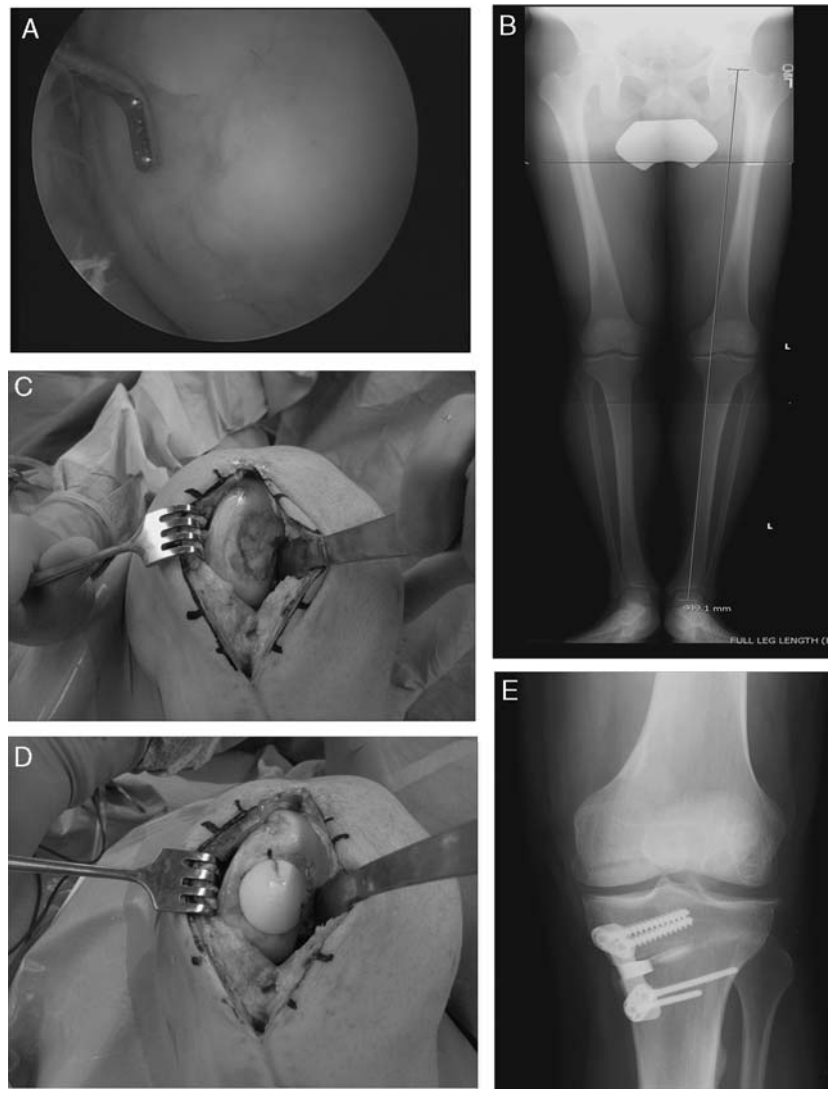

FIGURE 2. A 38-year-old active duty officer presented with a medial knee pain after undergoing a right autograft osteochondral transfer to his medial femoral condyle 10 years ago. His staging arthroscopy image (A) showed diffuse loss of articular cartilage with some maintenance at the site of osteochondral plugs. He underwent an open-wedge-high-tibial osteotomy for his varus deformity (B) and a subsequent allograft osteochondral transplantation for his diffuse chondral damage (C) using a single 30 mm plug (D). Final postoperative anteroposterior radiograph is shown (E).

FIGURE 3. A 22-year-old active duty soldier presented with posttraumatic chondral and bone loss (A) in his lateral femoral condyle after undergoing open reduction internal fixation of a distal femoral fracture. He had a valgus deformity (B and C) and was deficient of his lateral meniscus. He underwent a distal femoral osteotomy, lateral femoral condyle allograft osteochondral transplantation, and a lateral meniscus transplantation (D and E). His final alignment was symmetric and balanced (F and G).

irregular borders are debrided to a smooth surface while preserving the intact surrounding hyaline cartilage. The procedure is most commonly indicated for small, partial thickness cartilage lesions, patients with diffuse disease, or patients unwilling to comply with the rehabilitation restrictions associated with cartilage restoration procedures. Patients are able to progress through physical therapy without weight-bearing or range of motion restrictions and resume full activity within a few weeks.

Fixation

Fixation of osteochondral defects are most commonly indicated for OCD lesions and acute traumatic osteochondral fractures larger than 1 cm. The fragment must have adequate subchondral bone to allow osseous union. Fixation is indicated for International Cartilage Repair Society OCD II to IV lesions (Table 2). The fragment is assessed with a probe arthroscopically. If the fragment is unstable with probing, there is sufficient subchondral bone on the fragment, and anatomic reduction can be achieved, internal fixation is performed arthroscopically assisted or open.

Osteochondral fixation of unstable fragments begins with preparation of the fragment and the recipient site. An arthroscopic shaver, curette, or rasp is used to remove nonviable tissue. If the fragment has an intact hinge, it can often be reduced arthroscopically with a probe. Lesions with excessive bone loss may require bone grafting to ensure anatomic alignment of the articular surface. Often accessory portals or small open arthrotomies are necessary for fragment access, reduction, and fixation. With the fragment reduced provisional stabilization can be achieved with the guidewire of the cannulated fixation device (Figs. 4A–C). If the fragment is large enough, 2 screws will provide superior rotational stability. Fluoroscopic guidance is necessary to avoid crossing the physis in skeletally immature patients.

TABLE 2. ICRS Classification for OCD Lesions

ICRS OCD Classification	
ICRS OCD I	Stable, continuity: softened area covered by intact cartilage
ICRS OCD II	Partial discontinuity, stable on probing
ICRS OCD III	Complete discontinuity that are not yet dislocated (“dead in situ”)
ICRS OCD IV	Dislocated fragment, loose fragment within the bed, or empty bed

*Subgroup B for ICRS OCD I-IV indicates defects >10 mm in depth.

ICRS indicates International Cartilage Repair Society; OCD, osteochondritis dissecans.

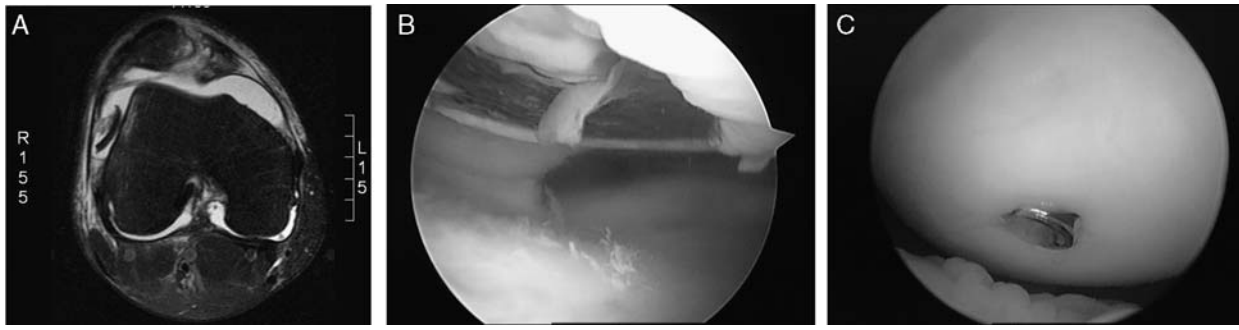

FIGURE 4. A, Axial magnetic resonance image showing displaced osteochondral fragment from lateral femoral condyle in 20-year-old lacrosse player who sustained a traumatic patella dislocation. B, Arthroscopic image of the lateral femoral condyle defect before debridement and fixation of lesion (through lateral parapatellar arthrotomy). C, Headless screw visible at the time of screw removal (12 wk).

For stable, nondisplaced symptomatic OCD lesions, drilling into the subchondral bone with a 0.062 Kirschner wire can stimulate a healing response. If the fragment is stable but MRI shows fluid tracking around the lesion, in-situ fixation can be performed with a headless compression screw or a cannulated screw. Hardware is removed 8 to 12 weeks after fixation. Bioabsorbable implants and osteochondral plugs may be used to obviate the need for hardware removal.[6] Postoperative rehabilitation involves continuous passive motion (CPM) use for 6 hours per day for 6 weeks. The patient is nonweight bearing in a brace when not participating in therapy.

Marrow Stimulation

Marrow stimulation is a comparatively simple and cost-effective technique to generate fibrocartilage formation in a chondral defect that can be used almost anywhere in the tibiofemoral articulation. Postoperative care requires patient compliance with rehabilitation protocols. Osteochondral drilling and microfracture techniques aim to release pluripotential cells and anabolic factors from the marrow to stimulate fibrocartilage fill of the defect.[16] This is a first-line treatment of grade 3 and 4 cartilage defects less than 2 cm^2. It can also be used on lesions larger than 3 cm in lower demand patients and on lesions on the tibial plateau because of technical difficulty of other procedures.

Arthroscopic shavers and curettes are used to remove unstable and damaged articular cartilage to subchondral bone. The goal is to create a well-defined lesion with vertical chondral "walls" perpendicular to the subchondral bone surface. Arthroscopic awls are placed 2 to 3 mm apart and to a depth of 4 to 5 mm.[17] With the arthroscopic pump turned off, blood and fat droplets can be seen extravasating from the microfracture sites (Figs. 5A, B). Postoperative rehabilitation involves nonweight bearing and CPM use for 6 hours per day for 6 weeks.

Autograft Osteochondral Transfer

Autologous osteochondral transfer involves the movement of cylindrical plugs of articular cartilage from nonweight-bearing areas of the knee to replace damaged cartilage in weight-bearing areas. It is a more technically demanding procedure, requires special instrumentation, has donor-site morbidity, and requires patients willing to comply with rehabilitation restrictions. However, it can be performed at the same setting of the initial surgery and does not incur the disease transmission risks or the costs associated with allograft osteochondral transplantation. It is indicated for grade 3 and 4 cartilage lesions up to 2.5 cm^2 in size involving the distal femoral condyles. Articular cartilage harvest sites include the superolateral lateral femoral condyle and intercondylar notch. "Mega-Osteoarticular Transfer System" procedures have been described for treating lesions up to 9 cm^2 with harvest from the posterior medial femoral condyle.[18]

The procedure is performed arthroscopically or with an associated small arthrotomy for access to the defect. After debridement of the lesion to subchondral bone, the site is drilled to prepare a uniform cylindrical recipient site. Subsequently, an adequate donor site is selected based on defect size. The preferred technique of the investigators is to harvest donor plugs from the lateral edge of the trochlea above the sulcus terminalis through a small arthrotomy and to place

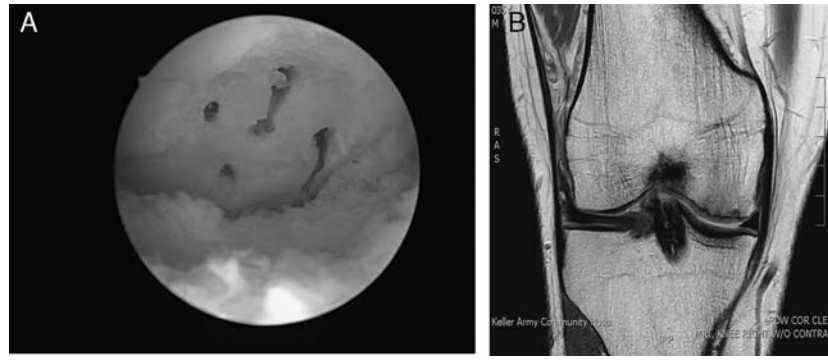

FIGURE 5. A, Medial femoral condyle articular defect treated with microfracture. B, Follow-up coronal magnetic resonance image showing fibrocartilage fill of the defect.

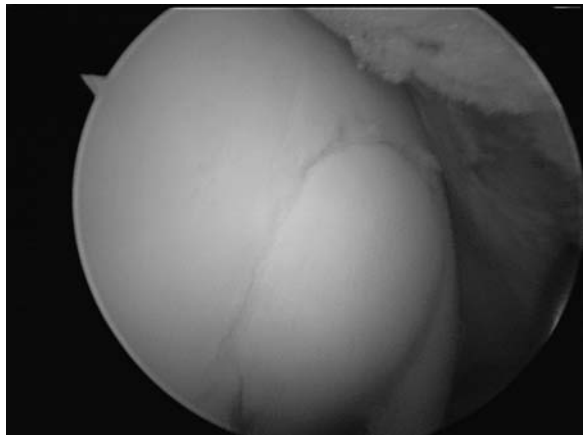

FIGURE 6. Arthroscopic view of the left knee medial femoral condyle lesion treated with single 10 mm autologous chondrocyte transfer placed arthroscopically.

the plugs arthroscopically (Fig. 6). Multiple cylindrical plugs (mosaicplasty) or a single cylinder can be placed in press-fit manner to fill a defect. Specialized sets are made by a number of manufacturers including MosaicPlasty (Smith Nephew, Andover, MA), Osteoarticular Transfer System (Arthrex, Naples, FL), and the Chondral Osseous Replacement System Osteochondral Defect Repair (Depuy Mitek, Westwood, MA). These systems assist with preparation of the recipient bed, harvest of the articular cartilage plug, and press-fit placement.

Grafts are placed flush with the surrounding articular cartilage. The surface contour of the defect and donor vary based on location, harvest site, and angle of drilling or harvest. In addition, because of the press-fit nature of fixation, the plugs cannot be rotated once impacted. As a result care must be taken to carefully evaluate the donor bed, cartilage contour, and plug for proper orientation during initial placement. Filling of the donor-site defects with bone graft or bone graft substitutes may reduce donor-site morbidity but has not been shown proven to alter outcomes. Postoperative rehabilitation involves nonweight bearing and CPM use 6 hours per day for 6 weeks.

Allograft Osteochondral Transplantation

Allograft osteochondral transplantation is technically similar to autograft but uses fresh cadaver distal femur for the donor plug. This enables grafting of defects larger than 2.5 cm^2, improved articular surface contour matching of large lesions, and is without the donor-site morbidity associated with autograft transfer. There is an increased financial cost associated with the allograft use, and it introduces the risk of disease transmission. Immune suppression is not required as transplanted chondrocytes are isolated by the cartilage matrix and are not exposed to immune surveillance.[19] Surgical coordination is more difficult in obtaining and implanting a fresh, size-matched condylar allograft within 4 weeks. Cold storage at 4°C showed cell viability of 65% to 90% suggesting implantation was possible up to 28 days after harvest.[20]

The procedure is performed similar to autograft osteochondral transfers, except that a donor harvest is not required. The investigators prefer to use an arthrotomy to ensure precision of plug alignment, orientation, and insertion (Figs. 2A–E). The donor plug can come from the same anatomic location improving the ease of contour matching. Although the articular cartilage is protected from immune surveillance, the subchondral bone on the allograft plug is a theoretic source of graft rejection. As a result, drilling the recipient site socket depth to 6 to 8 mm minimizes immunogenic donor bone. Press-fit fixation for large shallow allograft plugs can be supplemented with headless compression screws, resorbable implants, or cannulated metal screws. However, this is usually unnecessary. Postoperative rehabilitation includes nonweight bearing for 12 weeks to allow sufficient time for allograft incorporation. CPM use is recommended for 6 hours per day for 6 weeks.

ACI

ACI is a procedure involving the culturing of autogenous chondrocytes in vitro and implantation of these viable chondrocytes into a chondral defect. The procedure necessitates 2 operations and is currently financially burdensome. However, it is not associated with significant donor-site morbidity or risk of disease transmission. Carticel (Genzyme, Cambridge, MA) was the first cellular product licensed by the United States Food and Drug Administration and is approved for repair of symptomatic cartilaginous defects of the femoral condyles in patients with an inadequate response to earlier surgical procedures.[21] It is indicated for symptomatic, focal lesions up to 10 cm^2 in a patient willing to undergo the prolonged rehabilitation. Bipolar "kissing" lesions are a relative contraindication and lesions greater than 6 to 8 mm deep must have a concomitant bone grafting procedure.

The procedure is performed in 2 stages. The first procedure is a harvest of 200 to 300 mg of cartilage from the lateral femoral condyle or intercondylar notch. The cartilage biopsy is sent for processing and cellular expansion. The process takes a minimum of 6 weeks, but the cultured cells can be stored for up to 4 years with cryopreservation.[22] At least 6 weeks later, the cells are received from the laboratory and injected into the donor site. The defect is prepared to create vertical cartilage walls perpendicular to the subchondral bone. Care is taken not to violate the subchondral bone, which may allow influx of pluripotent cells and subsequent healing with fibrocartilage. A harvested periosteal patch from the proximal tibia or a collagen membrane is sewn over the cartilage defect with 6-0 suture in 5 mm intervals and sealed with fibrin glue. A small pocket on the superior aspect of the flap is left open for injection of the cultured cells. Final sutures and fibrin glue are placed to seal the lesion (Fig. 7).

Postoperative rehabilitation includes nonweight bearing for 12 weeks and CPM use 6 hours per day for 6 weeks. The

FIGURE 7. Autologous chondrocyte implantation performed on medial femoral condyle lesion (note good exposure provided by tibial tubercle osteotomy for treatment of concomitant patella lesion).

rehabilitation process is prolonged because of the time required to process the harvested chondrocytes and arrange reimplantation. Return to full activities is anticipated at 12 to 18 months after implantation. The length of rehabilitation and prolonged course away from full activities limits its use in competitive athletes and military personnel.

DISCUSSION

Clinical success of cartilage repair or restoration procedures is predicated on ligamentous stability, appropriate mechanical alignment, and treatment of meniscal pathology. Radiographic work-up can assist in surgical staging of the necessary procedures. The aforementioned pathology should be addressed before or during the definitive cartilage procedure. The various treatment options for chondral defects have reported variable success. Interpretation of the literature is challenging because of a lack of quality prospective studies, and the heterogeneous nature of these complex knee injuries and patients.

Microfracture is commony the first-line treatment offered to patients with a focal grade 3 or 4 cartilaginous defects smaller than 2 cm^2. Microfracture clinical results are better in patients younger than 30 to 40 years of age and with defects involving the femoral condyles.[23–25] Good-to-excellent results can be expected in 60% to 80% of patients with appropriately selected contained, isolated lesions of the femoral condyles in patients treated with microfracture.[17,23,26] Defect size larger than 2 to 4 cm^2 is associated with poorer clinical outcomes after microfracture.[24,27] Knutsen et al[23] reported no difference in radiographic, histologic, or clinical outcomes of focal condyle lesions treated with microfracture compared with ACI at 5 years. The investigators did note a trend toward more hyaline cartilage after ACI ($P = 0.08$). Microfracture is a safe first-line treatment of focal cartilaginous defects that can be performed in 1 setting, without donor-site morbidity, risk of disease transmission, need for special equipment or allograft specimens, no additional cost, and does not preclude the use of restorative procedures in the future.

ACI can be used to treat chondral defects up to 10 cm^2 without the risk of disease transmission associated with allograft osteochondral transplantation or the donor-site morbidity associated with autograft osteochondral transfer. The downside to ACI is the high financial cost associated with the procedure, the need for 2 surgical procedures, and the prolonged time required for rehabilitation. Recent advancements have obviated the need for periosteal patch harvest by covering the defect with a collagen membrane.[28] This reduces cartilage hypertrophy, minimizes operative time, and eliminates donor-site morbidity of periosteal harvest. Current advancements in ACI include matrix-induced ACI to allow all arthroscopic techniques, minimize operative time, faster rehabilitation, and allow access to lesions not amenable to open techniques.[29,30]

The theoretic benefit of ACI over microfracture is the development of hyaline-like cartilage rather than fibrocartilage. Studies have reported more hyaline-like cartilage compared with fibrocartilage but the long-term significance is unknown.[22] Clinical outcomes of primary ACI and as a reconstructive option after failed procedures report 70% to 90% good-to-excellent results.[22,31–33] Comparison of ACI with microfracture in 2 prospective trials showed conflicting results. Knutsen et al[23] reported satisfactory results in 77% of microfracture and ACI-treated lesions of the femoral condyles with a clinical or radiographic difference between the groups at 5-year follow-up. Kon et al[33] reported statistically better results with second generation ACI (Hyalograft C) in subjective and International Knee Documentation Committee scores at 5 years. Comparison of ACI with mosaicplasty also has had conflicting results reported in the literature. Bentley et al[34] showed 88% good results after ACI compared with 69% after mosaicplasty at 19-month follow-up for 4.66 cm^2 lesions. Dozin et al[35] reported equivalent results between ACI and mosaicplasty in a prospective randomized trial of 23 patients. ACI is likely indicated for lesions larger than 2 cm^2 when donor-site morbidity may outweigh advantages of native osteochondral plugs. A systematic review of ACI treatment of cartilage defects of the knee reported no significant difference in outcomes from ACI compared with other interventions and cited the need for long-term comparison trials of functional outcomes.[36]

Autograft osteochondral transfer allows placement of hyaline cartilage in cartilage defects rather than allowing the formation of fibrocartilage. The most significant down side is the donor-site morbidity; however, this procedure can be performed with a single surgery. Owing to limitations in available donor cartilage, autograft osteochondral transfer is limited to lesions smaller than 2.5 cm^2. Retrospective and prospective series have confirmed survival of hyaline cartilage at long-term follow-up and 76% to 90% good-to-excellent clinical results.[37] Pain, mechanical symptoms, and functional impairment can result from cartilage harvest.[38,39] A review of 200 patients with cartilage harvests from an asymptomatic knee for treatment of talus osteochondral lesions showed 10% of patients with Lysholm knee scores associated with a poor result.[39]

Allograft osteochondral transplantation eliminates donor-site morbidity associated with autograft harvest and allows treatment of larger chondral defects. This technique is the investigators' procedure of choice for all revision chondral procedures (Figs. 2A–E) or in cases with bone loss (Figs. 3A–G). The concern with use of allograft tissue is chondrocyte viability, graft incorporation, graft rejection, and risk of disease transmission. Methods of sterilization and storage have improved, thereby reducing the risk of disease transmission while maximizing chondrocyte viability. Fresh allografts stored at 4°C are most commonly used today and show a time-dependent decline in viability. Grafts are usually available 14 to 21 days after harvest, and the goal is to implant within 28 days of harvest.[40] The small window of graft implantation adds an additional scheduling difficulty to allograft use. Screening and sterilization minimize the risks of disease transmission with the risk of human immunodeficiency virus transmission estimated at 1 in 1.6 million before polymerase chain reaction testing.[40] Allografts do not require human leukocyte antigen or blood-type matching. The osseous component of the graft expresses surface antigens that can elicit an immune response. Processing and techniques to minimize osseous size limits the risk of immune reaction.

Cell viability has been shown in canine models to show time-dependent decline. Cell viability stored at 4°C was greater than 95% at 14 days, 75% to 98% at 21 days, and 65% to 90% at 28 days. Multiple studies have shown long-term chondrocyte viability after allograft transfer.[41,42] Fresh osteochondral allografts biopsied 2 years after implant showed 69% to 78% chondrocyte viability.[19] Five and 10-year survival of traumatic osteochondral distal femur defects in young active patients was reported at 95% and 85%, respectively. For focal cartilaginous defects, 84% of patients reported satisfaction with allograft osteochondral tissue transfer and 88% showed osseous incorporation radiographically.

CONCLUSIONS

Autograft osteochondral transfer, allograft osteochondral transplantation, ACI, fragment fixation, and microfracture have all showed successful clinical outcomes in the appropriately selected patient. There have been no long-term prospective functional outcome studies that have definitively proven superior results with any of the cartilage restoration procedures. Chondroplasty and lavage is a reasonable option in lower demand or older patients who want to avoid prolonged rehabilitation. Microfracture is a good first-line option in treating contained osteochondral defects smaller than 2 cm^2 of the femoral condyles or tibial plateau in high and low-demand patients willing to comply with rehabilitation. Autograft osteochondral transfer is an effective second-line option to provide native hyaline cartilage to a femoral condyle lesion and to avoid the expense, delay, and risks associated with ACI or allograft osteochondral transplantation. ACI or allograft transplantation have shown good clinical results treating defects larger than 2 cm^2 for patient willing to undergo prolonged rehabilitation and understand the risks of each procedure. The risks, additional procedures, and expense of these procedures are likely acceptable costs for high-demand patients.

REFERENCES

1. Curl WW, Krome J, Gordon ES, et al. Cartilage injuries: a review of 31,516 knee arthroscopies. *Arthroscopy* 1997;13:456–460.
2. Hjelle K, Solheim E, Strand T, et al. Articular cartilage defects in 1000 knee arthroscopies. *Arthroscopy* 2002;18:730–734.
3. Maletius W, Messner K. The effect of partial meniscectomy on the long-term prognosis of knees with localized, severe chondral damage. A twelve- to fifteen-year follow up. *Am J Sports Med* 1996;24:258–262.
4. Cole BJ, Pascual-Garrido C, Grumet RC. Surgical management of articular cartilage defects in the knee. *J Bone Joint Surg Am* 2009;91:1778–1790.
5. Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am* 2003;85-A(suppl 2):58–69.
6. 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.
7. Gudas R, Simonaityte R, Cekanauskas E, et al. 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.
8. Kirkley A, Griffin S, Richards C, et al. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder. *Arthroscopy* 1999;15:507–514.
9. Brittberg M, Peterson L. Introduction of an articular cartilage classification. ICRS Newsletter; 1998:5–8.
10. Griffin LY, Albohm MJ, Arendt EA, et al. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am J Sports Med* 2006;34:1512–1532.
11. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 1961;43-B:752–757.
12. Alford JW, Cole BJ. Cartilage restoration, part 2: techniques, outcomes, and future directions. *Am J Sports Med* 2005;33:443–460.
13. Sgaglione NA, Chen E, Bert JM, et al. Current strategies for nonsurgical, arthroscopic, and minimally invasive surgical treatment of knee cartilage pathology. *Instr Course Lect* 2010;59:157–180.
14. Williams RJ III, Brophy RH. Cartilage repair procedures: clinical approach and decision making. *Instr Course Lect* 2008;57:553–561.
15. Alford JW, Cole BJ. Cartilage restoration, part 1: basic science, historical perspective, patient evaluation, and treatment options. *Am J Sports Med* 2005;33:295–306.
16. Shapiro F, Koide S, Glimcher MJ. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. *J Bone Joint Surg Am* 1993;75:532–553.
17. Steadman JR, Rodkey WG, Briggs KK. Microfracture to treat full-thickness chondral defects: surgical technique, rehabilitation, and outcomes. *J Knee Surg* 2002;15:170–176.
18. Brucker PU, Braun S, Imhoff AB. Mega-OATS technique--autologous osteochondral transplantation as a salvage procedure for large osteochondral defects of the femoral condyle. *Oper Orthop Traumatol* 2008;20:188–198.
19. Czitrom AA, Keating S, Gross AE. The viability of articular cartilage in fresh osteochondral allografts after clinical transplantation. *J Bone Joint Surg Am* 1990;72:574–581.
20. Williams JM, Virdi AS, Pylawka TK, et al. Prolonged-fresh preservation of intact whole canine femoral condyles for the potential use as osteochondral allografts. *J Orthop Res* 2005;23:831–837.
21. Wood JJ, Malek MA, Frassica FJ, et al. Autologous cultured chondrocytes: adverse events reported to the United States Food and Drug Administration. *J Bone Joint Surg Am* 2006;88:503–507.
22. Brittberg M, Lindahl A, Nilsson A, et al. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med* 1994;331:889–895.
23. Knutsen G, Drogset JO, Engebretsen L, et al. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. *J Bone Joint Surg Am* 2007;89:2105–2112.
24. Knutsen G, Engebretsen L, Ludvigsen TC, et al. Autologous chondrocyte implantation compared with microfracture in the knee. A randomized trial. *J Bone Joint Surg Am* 2004;86-A:455–464.
25. Kreuz PC, Erggelet C, Steinwachs MR, et al. Is microfracture of chondral defects in the knee associated with different results in patients aged 40 years or younger? *Arthroscopy* 2006;22:1180–1186.
26. Mithoefer K, Williams RJ III, Warren RF, et al. The microfracture technique for the treatment of articular cartilage lesions in the knee: a prospective cohort study. *J Bone Joint Surg Am.* 2005;87:1911–1920.
27. Gudas R, Kalesinskas RJ, Kimtys V, et al. A prospective randomized clinical study of mosaic osteochondral autologous transplantation versus microfracture for the treatment of osteochondral defects in the knee joint in young athletes. *Arthroscopy* 2005;21:1066–1075.
28. Steinwachs M, Kreuz PC. Autologous chondrocyte implantation in chondral defects of the knee with a type I/III collagen membrane: a prospective study with a 3-year follow-up. *Arthroscopy* 2007;23:381–387.
29. Bartlett CS, DiFelice GS, Buly RL, et al. Cardiac arrest as a result of intraabdominal extravasation of fluid during arthroscopic removal of a loose body from the hip joint of a patient with an acetabular fracture. *J Orthop Trauma* 1998;12:294–299.
30. Wondrasch B, Zak L, Welsch GH, et al. Effect of accelerated weightbearing after matrix-associated autologous chondrocyte implantation on the femoral condyle on radiographic and clinical outcome after 2 years: a prospective, randomized controlled pilot study. *Am J Sports Med* 2009;37(suppl 1):88S–96S.
31. Peterson L, Minas T, Brittberg M, et al. Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. *Clin Orthop Relat Res* 2000;374:212–234.
32. Zaslav K, Cole B, Brewster R, et al. A prospective study of autologous chondrocyte implantation in patients with failed prior treatment for

articular cartilage defect of the knee: results of the Study of the Treatment of Articular Repair (STAR) clinical trial. *Am J Sports Med* 2009;37:42–55.

33. Kon E, Gobbi A, Filardo G, et al. Arthroscopic second-generation autologous chondrocyte implantation compared with microfracture for chondral lesions of the knee: prospective nonrandomized study at 5 years. *Am J Sports Med* 2009;37:33–41.
34. 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.
35. Dozin B, Malpeli M, Cancedda R, et al. Comparative evaluation of autologous chondrocyte implantation and mosaicplasty: a multicentered randomized clinical trial. *Clin J Sport Med* 2005;15:220–226.
36. Wasiak J, Clar C, Villanueva E. Autologous cartilage implantation for full thickness articular cartilage defects of the knee. *Cochrane Database Syst Rev* 2006;3:CD003323.
37. Hangody L, Vasarhelyi G, Hangody LR, et al. Autologous osteochondral grafting—technique and long-term results. *Injury* 2008;39(suppl 1): S32–S39.
38. LaPrade RF, Botker JC. Donor-site morbidity after osteochondral autograft transfer procedures. *Arthroscopy* 2004;20:e69–e73.
39. Paul J, Sagstetter A, Kriner M, et al. Donor-site morbidity after osteochondral autologous transplantation for lesions of the talus. *J Bone Joint Surg Am* 2009;91:1683–1688.
40. Lattermann C, Romine SE. Osteochondral allografts: state of the art. *Clin Sports Med* 2009;28:285–301, ix.
41. Gross AE, Kim W, Las Heras F, et al. Fresh osteochondral allografts for posttraumatic knee defects: long-term follow up. *Clin Orthop Relat Res* 2008;466:1863–1870.
42. Maury AC, Safir O, Heras FL, et al. Twenty-five-year chondrocyte viability in fresh osteochondral allograft. A case report. *J Bone Joint Surg Am* 2007;89:159–165.

