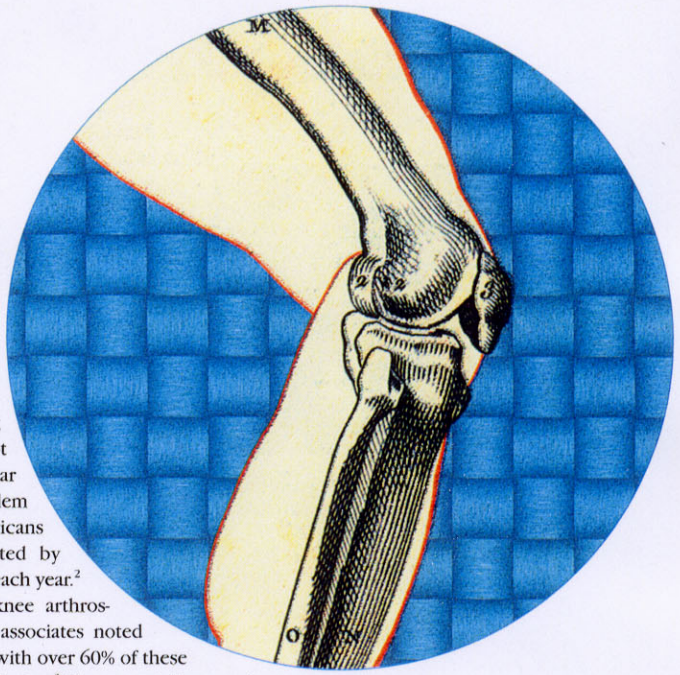
Chondral Injuries of the Knee

A Contemporary View of Cartilage Restoration

Brian J. Cole, MD, MBA
Assistant Professor of Sports Medicine
Medical Director
Rush Cartilage Restoration Center

Brian Cohen, MD
Sports Medicine Fellow
Section of Sports Medicine

Rush–Presbyterian–St. Luke's Medical Center
Chicago, Illinois



Physicians have known for over 250 years that articular cartilage damage is a "troublesome thing and once destroyed, it is not repaired."[1] Injury to the articular cartilage of the knee is a serious problem affecting an estimated 900,000 Americans annually, with high-grade lesions treated by more than 200,000 surgical procedures each year.[2] In a retrospective review of 31,516 knee arthroscopies over a 4-year period, Curl and associates noted articular damage in 63% of the patients, with over 60% of these having a grade III or grade IV chondral lesion.[3] The natural history of chondral injury is not well defined, but once patients become symptomatic from these lesions, progression is likely.

Focal chondral defects of the femur make up a specific subset of articular cartilage injuries (Figure 1). Reports have shown that even unipolar, unicompartmental articular cartilage injuries have a greater than 50% chance of becoming symptomatic with demonstrable joint-space narrowing.[4] The clinical course is multifactorial and dependent on lesion-specific and patient-specific factors. Lesion size, location, depth, chronicity, and response to previous treatment are important considerations. Associated comorbidities such as cruciate deficiency, meniscal damage, limb malalignment, and obesity are also factors to consider in evaluation and treatment. Treatment for symptomatic lesions is primarily surgical. A full understanding of the patient's level of impairment allows the surgeon to choose an appropriate treatment option.

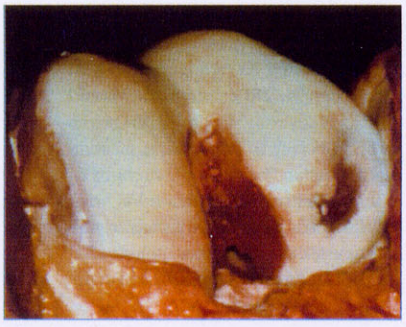


Figure 1.

Arthroscopic example of a symptomatic full-thickness chondral defect of the medial femoral condyle.

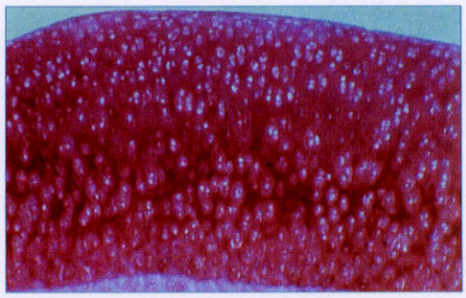


Figure 2a.

Organized architecture of normal articular cartilage—high-power light microscopy, hematoxylin & eosin stain.

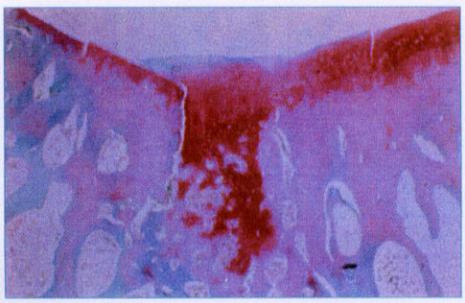


Figure 2b.

Disorganized architecture of fibrocartilage following microfracture technique—high-power light microscopy, hematoxylin & eosin stain.

Table 1. Modified International Cartilage Repair Society Chondral Injury Classification System

Grade of Injury	Description
Grade 0	Normal
Grade I	Superficial fissuring
Grade II	<1/2 of cartilage depth
Grade III	>1/2 of cartilage depth to subchondral plate
Grade IV	Osteochondral lesion through subchondral plate
Osteochondritis dissecans	Stability Continuity Depth (relative to 10 mm)

Pathophysiology

Despite its relatively unremarkable appearance (Figure 2a), articular cartilage has a unique ability to provide a low-friction surface and survive repetitive loading in compression, shear, and tension for many decades. It is avascular, aneural, alymphatic, and contains a single cell type, the chondrocyte. Its lack of vascularity, high matrix-to-cell ratio, and lack of a local undifferentiated cell pool leads to its limited capacity to regenerate following injury.

Classification of chondral injuries focuses on the amount and depth of the cartilage lesion (Table 1). Regardless of the type of injury, without intervention there is no chance for articular cartilage to regenerate normal hyaline cartilage. Violation of the subchondral plate will, however, expose the damaged area to progenitor cells residing within the subchondral bone, thereby leading to fibrocartilage repair tissue (Figure 2b). However, this tissue is biologically and biomechanically inferior to hyaline cartilage and demonstrates a preponderance of Type I collagen rather than the normally abundant Type II collagen. The relationship between focal cartilage injury and the development of degenerative arthritis is still under investigation. The similar biologic, mechanical, and macroscopic features indicate that "both conditions may be part of a continuum of joint deterioration."[2] This assumed relationship is the primary rationale for early intervention in symptomatic patients.

Patient Evaluation

Cartilage injuries can occur in isolation or in association with other intra-articular pathology. The accurate diagnosis of a symptomatic focal chondral defect requires that the evaluator maintain a high index of suspicion for this pathologic entity, especially in the presence of concomitant pathology such as meniscal or ligament tears. Symptoms may be subtle but often include localized pain, catching, swelling, and giving-way. A thorough history should elicit the mechanism of injury, previous injuries, and symptom-provoking activities. A complete physical examination is essential to evaluate for concomitant pathology that would alter the treatment plan (Table 2).

Diagnostic imaging is required and should begin with a standard weight-bearing, anteroposterior (AP) radiograph of both knees in full extension, a non-weight-bearing 45-degree flexion lateral view and an axial view of the patellofemoral joint. Additionally, a 45-degree flexion weight-bearing posteroanterior (PA) radiograph can help identify subtle joint-space narrowing that traditional extension views may fail to uncover.[5] Special studies such as a long-cassette mechanical axis view or a magnetic resonance imaging (MRI) evaluation should be done as needed.

If joint-space narrowing is present on the 45-degree flexion weight-bearing PA radiograph, an MRI is rarely necessary. Generally, MRI examination should be reserved for difficult cases in which the diagnosis remains unknown, especially in the setting of completely normal radiographs. The greatest strength of the MRI is its ability to evaluate the subchondral bone (ie, osteochondral fractures, osteonecrosis, and osteochondritis dissecans). MRI techniques include 2-D fast-spin-echo and 3-D fat suppression with and without intra-articular gadolinium.[6]

Nonsurgical Management

Nonsurgical management is largely ineffective in symptomatic patients and should be reserved for relatively low-demand patients, patients wishing to avoid or delay surgery, and patients with advanced, degenerative osteoarthritis considered inappropriate for articular cartilage restoration procedures.[2]

Table 2. Components of a Comprehensive Musculoskeletal Physical Examination[2]

Alignment
Varus (bow-legged)
Valgus (knocked-kneed)
Gait
Antalgic
Flexed-knee
Recurvatum (hyperextended)
Compensatory
Thrust
Varus (lateral)/Valgus (medial)
Swelling
Soft tissue
Effusion
Ligament laxity
Anteroposterior (ACL/PCL)
Medial-lateral (MCL/LCL)
Range of motion
Strength/muscle atrophy
Specific compartments
Patellofemoral
Tibiofemoral
Meniscus
Joint-line tenderness
Provocative maneuvers
Related joints
Spine
Hips
Feet
Neurovascular evaluation

Key

ACL	anterior cruciate ligament
LCL	lateral collateral ligament
MCL	medial collateral ligament
PCL	posterior cruciate ligament

Treatment options include nonsteroidal anti-inflammatory drugs (NSAIDs), judicious use of corticosteroid injections, and/or use of oral or injectable chondroprotective agents. Alternatives include: activity modification with avoidance of high-impact activities; physical therapy focusing on muscle strengthening and hamstring flexibility; and use of a knee sleeve or an unloader brace to improve proprioception or unload diseased cartilage, respectively.

Although the natural history of a focal chondral lesion is poorly understood, the symptomatic lesion is likely to lead to disease progression, which would make future surgical treatment options more complicated. When surgical intervention is indicated, a clear definition of lesion size, depth, and location is required to determine the procedure of choice. Concomitant management of associated conditions such as malalignment, ligament insufficiency, and/or meniscal injury is essential for a successful outcome.

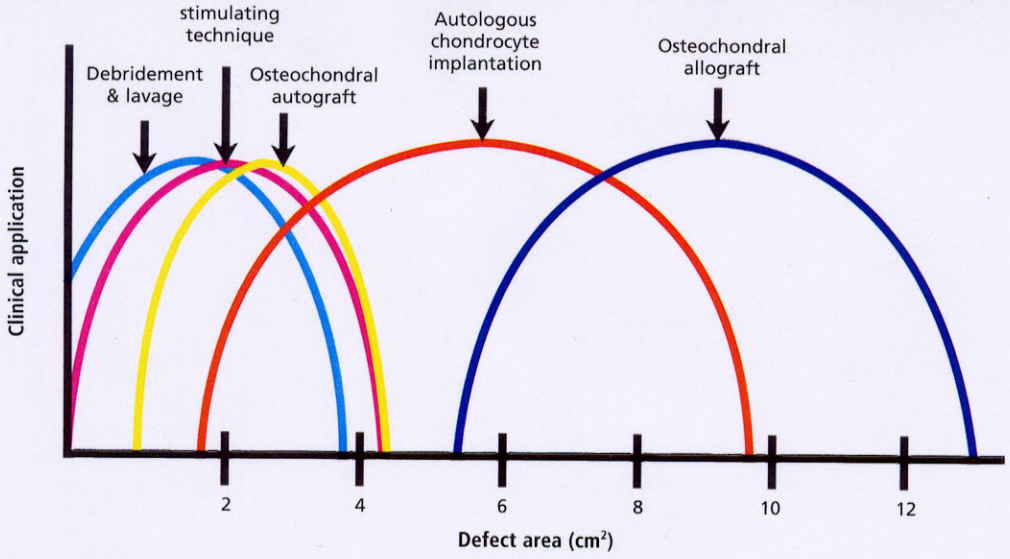


Figure 3. Phase shift diagram emphasizing overlapping indications for treatment options when size alone is considered.

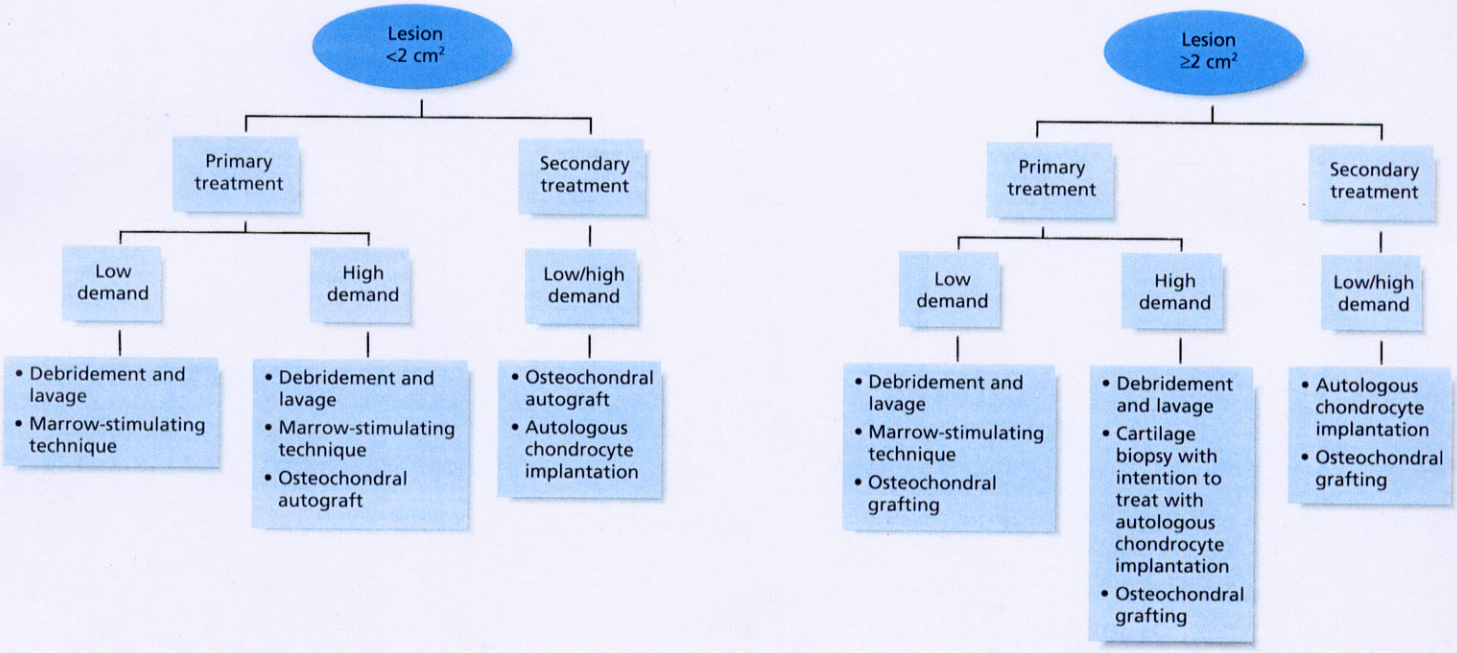


Figure 4. Composite treatment algorithm for management of the symptomatic focal cartilage defect of the femur.

Surgical Management

The principal goals for surgical management of the symptomatic chondral defect are to reduce symptoms, improve joint congruence by restoring the joint surface with the most normal tissue (ie, hyaline cartilage) possible, and to prevent additional cartilage deterioration. Although the need for surgical management is based on the patient's history, physical examination, and diagnostic studies, only knee arthroscopy will provide definitive information regarding the lesion's characteristics (ie, location, size, depth, degree of containment) and associated injuries (ligamentous and/or meniscal).

Procedures are classified by their relative ability to promote and restore the damaged articular surface. Surgical management traditionally follows a treatment algorithm that is directed principally by lesion size (ie, relative to 2 cm^2). Considering size alone, however, is insufficient to guide treatment due to overlapping indications for many of the available treatment options (Figure 3). In addition to lesion size, assessing the patient's current and desired activity level, symptom intensity, and response to previous treatment is helpful to compartmentalize treatment options into an all-inclusive treatment algorithm (Figure 4).

Based upon their anticipated outcome, it is helpful to define treatment options as being palliative, reparative, or restorative (Table 3, page 74). First-line treatment for smaller injuries in lower-demand patients with limited symptoms can be treated effectively with **palliative** procedures such as *debridement and lavage*. Relief, however, may be incomplete and short-lived. Mid-sized lesions in patients with moderate symptoms can be treated with a **reparative** procedure using a *marrow-stimulating technique* (ie, drilling, abrasion arthroplasty, or microfracture) in an effort to promote a fibrocartilage healing response. Results in larger lesions in higher-demand patients, however, are generally less favorable and shorter-lived, independent of any prior treatments rendered. Larger defects, especially in higher-demand patients with significant symptoms who have failed less aggressive primary treatment options, are most effectively treated with a **restorative** treatment option such as *autologous chondrocyte implantation (ACI)* or *osteochondral grafting*.

Palliative Treatment: Debridement and Lavage

Arthroscopic debridement and lavage is best performed as a first-line surgical approach for smaller lesions (0.5-3 cm^2) in lower-demand patients with

Table 3. Surgical Treatment Options for Symptomatic Focal Cartilage Defects*

Lesion	Treatment	Treatment Outcome	Rehabilitation	Comments	Complications	Technical Difficulty	Relative Costs
Primary Treatment							
<2 cm^2	Debridement and lavage	Palliative	Early weight-bearing, ROM, and strengthening	Ideal for low-level symptoms; short-term symptomatic relief	Rare, persistent pain, stiffness[9]	Low	Low
	Marrow-stimulating technique	Reparative	Prolonged, protected weight-bearing and return to activities (4-6 mo)	Ideal for smaller femoral condyle or trochlear lesions; intermediate-term relief	Rare, persistent pain, stiffness[9]; progressive cartilage degeneration	Moderate	Low
	Osteochondral autograft	Restorative	Short-term, protected weight-bearing and return to activities within 3-4 mo	Probably as good if not better than MST; potentially long-term relief	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions	High	Moderate
>2 cm^2	Debridement and lavage†	Palliative	Early weight-bearing, ROM, and strengthening	Ideal for low-level symptoms; short-term symptomatic relief	Rare, persistent pain, stiffness[9]	Low	Low
	Marrow-stimulating technique	Reparative	Prolonged, protected weight-bearing and return to activities (4-6 mo)	Less success in larger lesions; good choice for symptomatic relief in low-demand, low-level symptomatic individuals; intermediate-term relief possible	Rare, persistent pain, stiffness[9]; progressive cartilage degeneration	Moderate	Low
	Osteochondral autograft	Restorative	Prolonged, protected weight-bearing and return to activities (3-4 mo)	Larger lesions with increased donor site morbidity; variable results	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions	High	Moderate
	Osteochondral allograft	Restorative	Prolonged, protected weight-bearing and significant delay until return to activities (6-8 mo)	Larger lesions with significant bone loss; small concern for disease transmission and allograft availability; potentially long-term relief	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions; potential for disease transmission, immunologic response	High	High
Secondary Treatment							
<2 cm^2	Osteochondral autograft	Restorative	Short-term, protected weight-bearing and return to activities within 3-4 mo	Probably as good if not better than MST; potentially long-term relief	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions	High	Moderate
	Autologous chondrocyte implantation	Restorative	Prolonged, protected weight-bearing and significant delay until return to activities (8-12 mo)	Significant improvement with potentially long-term relief	Occasional stiffness, periosteal complications, delamination[21]	High	High
>2 cm^2	Osteochondral autograft	Restorative	Prolonged, protected weight-bearing and significant delay until return to activities (3-4 mo)	Larger lesions with increased donor site morbidity; variable results	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions	High	Moderate
	Osteochondral allograft	Restorative	Prolonged, protected weight-bearing and significant delay until return to activities (6-8 mo)	Larger lesions with significant bone loss; small concern for disease transmission and allograft availability; potentially long-term relief	Improper harvest and implantation, contour mismatch, donor morbidity, plug failure, hemarthrosis, effusions; potential for disease transmission, immunologic response	High	High
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*Procedure selection will ultimately depend on the patient's symptom level, age, expectations, activity level, coexisting pathology, previous treatments, and extent and location of lesion.
†Consider concomitant cartilage biopsy with intention to treat with autologous chondrocyte implantation as a secondary treatment.

Key

ACI	Autologous chondrocyte implantation
MST	Marrow-stimulating technique
ROM	Range of motion

recurrent effusions are present. Success is achieved by improving articular surface congruity, while eliminating debris and inflammatory mediators.[7] In relatively young or active individuals with moderate symptoms and larger lesions (>2 cm^2), results have been less promising, demonstrating only temporary, symptomatic relief.[8,9] Thermal debridement of superficial articular cartilage injuries is being investigated; some evidence suggests that, when it is used as an adjunct to mechanical shaving, articular contouring can be achieved. However, the optimal depth of penetration and clinical correlation with macroscopic and microscopic alterations are not defined.[10,11]

Reparative Treatment: Marrow-Stimulating Techniques

Marrow-stimulating techniques include subchondral drilling, abrasion arthroplasty (Figure 5a), and microfracture (Figure 5b). The objective of these procedures is to expose the chondral defect to the pluripotential marrow stem cells that reside below the subchondral bone and have the capacity to form fibrocartilage in the base of the defect (Figure 6). Studies have shown, however, that fibrocartilage is unable to "function properly in a high-stress environment with load bearing and may actually lead to further cartilage degeneration and osteoarthritis."[12]

Marrow-stimulating techniques are recommended for smaller lesions (<2 cm^2) in active patients with no more than moderate symptoms, or for larger lesions (>2 cm^2) in lower-demand patients with mild symptoms. Results indicate that 60% to 75% of patients with smaller lesions will have symptomatic relief for up to 3 years, or longer in some cases, after treatment with a marrow-stimulating technique.[13-15] Results are less predictable and less successful for larger defects or lesions in the trochlear groove and tibial condyle.

Complications are rare and mimic those seen following arthroscopic debridement and lavage. Progressive cartilage degeneration and recurrent symptoms are the most common complications, and close postoperative monitoring of patients is required. Microfracture is favored over subchondral drilling and abrasion arthroplasty because it is less destructive to the subchondral bone.

Restorative Treatment

Restorative procedures, such as ACI and osteochondral grafting, succeed by re-establishing normal articular congruity with mechanically stable hyaline or hyaline-like cartilage. Due to their complexity and generally higher cost, they are best reserved for higher-demand patients, patients with significant symptoms, and patients who have failed prior palliative and reparative procedures.

Autologous Chondrocyte Implantation.—ACI biologically resurfaces focal chondral defects with "hyaline-like" cartilage, which is believed to be biologically and mechanically superior to fibrocartilage.[16] ACI is indicated in higher-demand patients with symptomatic deep grade III or IV lesions of the femur between 2 cm^2 and 10 cm^2 (Figure 7a). It is most commonly performed as a secondary treatment after previous treatment failure. It is a staged, restorative procedure, requiring a cartilage biopsy at the index procedure from a minor load-bearing area of the knee joint (Figure 7b). At the follow-up procedure, an arthrotomy is performed, the defect is meticulously prepared, and cultured chondrocytes are reinjected beneath a periosteal patch sewn with multiple interrupted sutures and secured with fibrin glue (Figure 7c). Over time, hyaline-like cartilage will fill the defect (Figure 7d,e).

Figure 5a.

Arthroscopic view of abrasion arthroplasty technique.

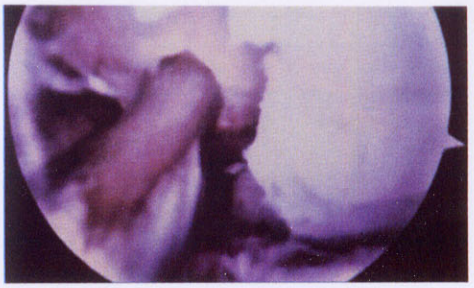

Figure 5b.

Arthroscopic view of microfracture technique.

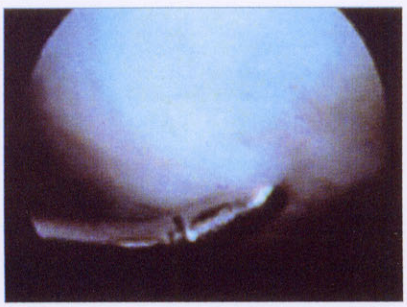

Figure 6.

Arthroscopic view of mature fibrocartilage following marrow-stimulating technique.

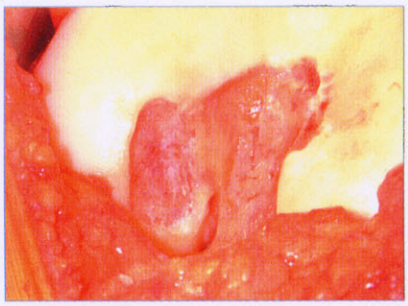

Figure 7a.

Symptomatic full-thickness focal chondral defect prepared for autologous chondrocyte implantation.

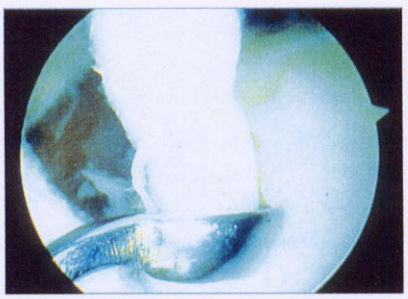

Figure 7b.

Articular cartilage biopsy for future autologous chondrocyte implantation.

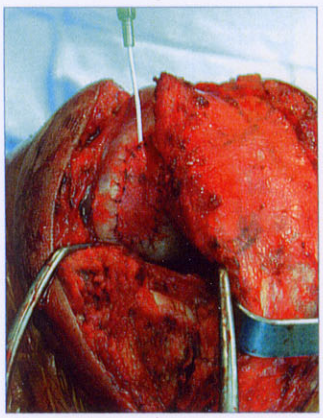

Figure 7c.

Injecting cells beneath periosteal patch.

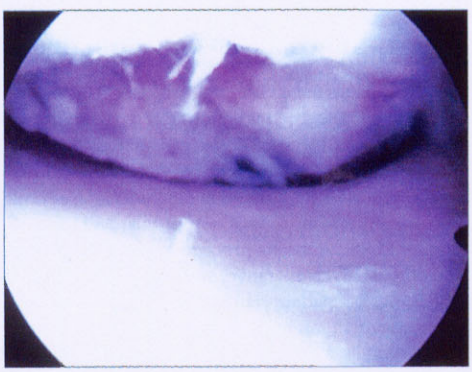

Figure 7d.

Arthroscopic view of symptomatic medial femoral condyle defect indicated for autologous chondrocyte implantation after failed abrasion arthroplasty.

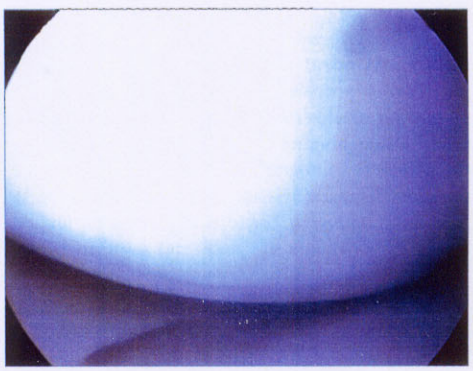

Figure 7e.

Second-look arthroscopic view of same defect 18 months after autologous chondrocyte implantation.

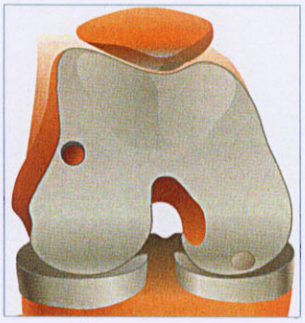

Figure 8.

Schematic of osteochondral autograft transplant system.

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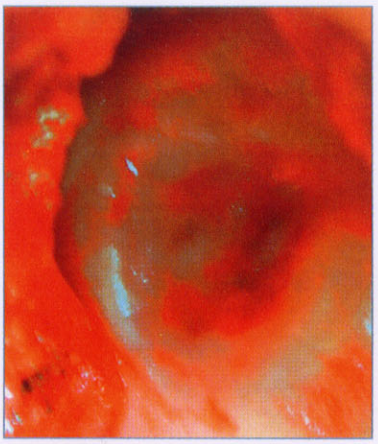

Figure 9a.

8-cm^2 OCD lesion of the lateral femoral condyle indicated for osteochondral allograft procedure.

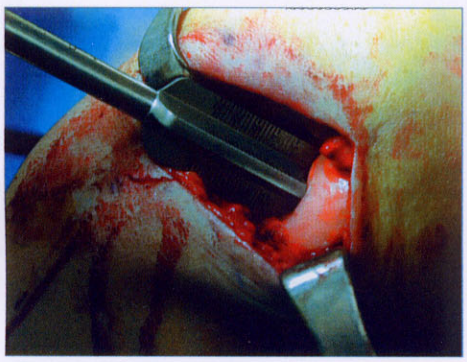

Figure 9b.

Reaming of defect in preparation for shell allograft.

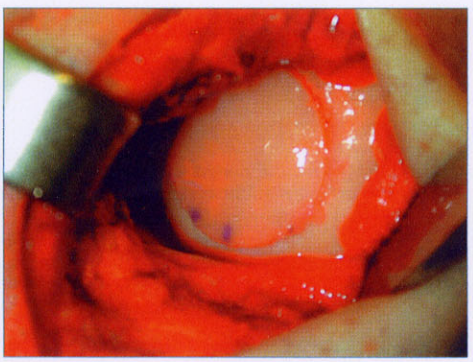

Figure 9c.

Allograft press-fit within defect.

Follow-up studies indicate that one can expect a greater than 80% rate of good-to-excellent results following ACI when it is appropriately performed.[17,18] The location of the injury plays a role in the success of the procedure, with clinical improvement seen in >90% for isolated femoral condylar lesions, with follow-up as long as 9 years, and as low as 60% for lesions of the patella.[17,19] In general, it is believed that "for carefully selected patients having full-thickness cartilage defects in the knee, ACI provides substantial improvement in quality of life and is very cost-effective."[20]

Osteochondral Grafting. Osteochondral grafting restores articular congruity by transplanting a composite of subchondral bone and hyaline cartilage.[21] Osteochondral tissue is obtained from either the patient (ie, autograft) or from a cadaveric source (ie, allograft) made available as a fresh or prolonged-fresh graft. Autograft tissue is restricted by limited availability of donor site areas and associated morbidity, and thus, only relatively small defects are appropriately treated with this option.

Osteochondral autograft transplantation is indicated in patients with traumatic, focal chondral defects (1-3 cm^2) with limited subchondral bone loss (<6 mm) (Figure 8).[22] Although long-term follow-up is forthcoming, results at 5 years suggest that this treatment is better than marrow-stimulating procedures for similarly sized lesions.[23]

Osteochondral allograft transplantation is indicated for larger lesions (>2 cm^2) with associated bone loss. The procedure relies on precision instrumentation of size-matched donor tissue to effectively restore articular congruity (Figures 9a-9c). Fresh osteochondral tissue demonstrates greater than 60% donor chondrocyte viability at biopsy.[24] Clinical outcomes indicate good to excellent results in excess of 80% of patients treated for unipolar, unicompartmental lesions.[25]

Conclusion

Symptomatic focal chondral defects of the articular surface of the knee are a complex clinical problem because of the inability of articular cartilage to initiate any clinically appreciable healing response. When indicated, treatment should ideally prevent defect progression, reduce symptoms, and restore function. Indications to proceed with options considered palliative, reparative, or restorative are evolving. Typically, patient- and lesion-specific factors guide treatment. An understanding of the indications and outcomes allows the surgeon to appropriately match the treatment option to the patient's level of impairment, optimizing the opportunity for a successful and uncomplicated recovery. ❖

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