Patient Evaluation and Surgical Decision Making

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INTRODUCTION

Treatment of damaged articular cartilage is a challenging process through which the patient and physician navigate together. Because articular cartilage lesions have limited capacity to heal themselves, identifying the optimal treatment plan for a particular type of cartilage lesion is critical to achieve symptom reduction and improvements in function.

Choosing the optimal treatment is a particular process, as each treatment option embodies biologic, biomechanical, and technical factors. Additionally, with consideration to relevant comorbidities, the specific treatment option must appropriately be matched to the patient and the general condition of the knee. Finally, the patient's psychosocial disposition and the pathoanatomy leading to their subjective complaints and objective dysfunction must also be considered to appreciate the entire patient picture. Treatment selection is, therefore, predicted on patient- and knee-specific articular cartilage factors.

The purpose of this article is not to review the specific techniques, indications, or results of available treatment options, which are well described elsewhere,[11,15-18,49] but rather to present a rational, patient- and knee-specific demand-match approach to the treatment of articular cartilage lesions.[10]

CLINICAL EVALUATION

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Independent of the articular cartilage lesion, patient-factors influence the treatment recommendations and determine the likelihood of achieving a positive outcome. Effective physician-patient communication should emphasize a mutual understanding of potential outcomes and establish realistic expectations. The impact of psychosocial parameters cannot be overemphasized.

Minas et al[42] reported that the strongest predictor of success following autologous chondrocyte implantation was the "overall sense of well being" as determined by the Short Form-36 score. Although this implies that poor outcomes might be expected in poorly motivated patients (ie, workers' compensation cases), at least one published study suggests that results are not entirely discouraging in that population.[55] Nevertheless, the surgeon needs to formulate an assessment of what the patient can expect as an outcome in terms of reductions in pain and improvements in function. It is important to note that outcomes are dependent on surgeon and patient effort, ie, restoring a grossly appearing normal articular surface is not synonymous with normal biomechanical function. The restored surface may not be able to tolerate excessive overload. Thus, for example, joint overload that is due to an excessive body mass index must be minimized by addressing excessive body weight prior to initiating surgical intervention.

HISTORY

Patients with focal chondral lesions present in a variety of ways. Most commonly, though, patients present with a chief complaint of pain in the respective compartment (ie, medial, lateral, or anterior). Direct weight bearing may aggravate tibiofemoral symptoms whereas stair climbing and squatting would typically increase patellofemoral symptoms. Other common symptoms include localized pain, effusion, catching and occasional-

ly locking, or mechanical symptoms. Global or atypical pain patients should be thoroughly evaluated for other causes of pain (eg, chronic regional pain syndrome) even if they have a documented articular lesion, because, again, "successful" treatment may not fulfill patient expectations in alleviating pain, ie, patient-rated success is the ultimate goal, not simply lesion restoration success.

Acute injuries may be associated with a twisting or shearing mechanism with or without blunt trauma. The injuries that result in acute chondral defects often are immediately symptomatic. Alternatively, those which injure or kill chondrocytes leading to the inability to maintain the matrix over time (ie, injuries with extensive "bone bruising" on magnetic resonance imaging [MRI]) may initially have symptom resolution as the soft-tissue pain subsides, only to have gradual return of symptoms as the matrix deteriorates leading ultimately to failure of the articular surface. Not uncommonly, acute articular cartilage injuries are associated with concomitant meniscus and ligament tears.[35] As such, it is important to consider the potential of "discovering" a significant articular cartilage lesion whenever an associated meniscal or ligament disruption is present. This point is important in the preoperative counseling of patients who might have an unsuspected articular cartilage lesion in need of treatment and should be a part of a thorough preoperative informed consent process.

Focal lesions may also present in a subacute or chronic time frame often associated with prolonged exposure to abnormal biomechanics and microtrauma. For example, trochlear lesions are not uncommon in patients with normal patellofemoral alignment due to prolonged participation in high patellofemoral loading sports such as basketball or competitive skiing. Tibiofemoral deterioration often is seen following meniscectomy and may begin as a relatively localized area of cartilage deterioration only to progress to bipolar change over variable periods of time often dependent on other factors such as malalignment. Finally, adults with osteochondritis dissecans can cope with minor symptoms for years and then present with pain and effusions as the fragment dislodges. In this case, treatment may only require loose body removal as the defect itself may remain relatively asymptomatic when lesion dimensions are in the "small category." Thus, knowledge of the timeline of articular cartilage deterioration may aid in evaluating the relative urgency and need for cartilage restoration.

It is imperative to relate the patient's pain to the cartilage pathology. Although obvious to most surgeons, it is not obvious to most patients that joint pain may be, in fact, not directly related to articular cartilage pathology ascertained from imaging studies or following arthroscopic evaluation. The relationship of pain to the extent of articular cartilage injury is at best indirect. As articular cartilage lacks a nerve supply, the pain originates from either the subchondral region (bone, nerve, and vascular network) or from the soft tissues stimulated by the local biochemical/mechanical effects of flaps or desquamated cartilage. As a corollary, the intensity of pain does not predict the extent or severity of articular cartilage damage. In some patients, a subconscious decrease in their activity levels allow them to better "tolerate" the pain. Therefore, it is important to include the previous, as well as current, activity level and their association with defect-related symptoms in the history. It may be helpful for patients to temporarily return to their previous (and desired) activity level and maintain a diary of their symptoms, which can be discussed at subsequent office visits. Finally, obtaining a family history for osteoarthritis is important as the prognosis may be more guarded due to a genetic diathesis for cartilage degeneration despite appropriate treatment.

PHYSICAL EXAMINATION

Often objective findings are absent and complaints of pain are the primary reason for patients to seek treatment. Typically, range of motion is preserved. Objectively, patients may present with effusions and focal tenderness in the involved compartment. Global or diffuse pain should serve as a warning that the isolated cartilage lesion is not the major problem. A standard, thorough physical assessment of the knee is essential to evaluate for coexisting pathology such as ligament or meniscal deficiency or mechanical axis malalignment.

The physical examination will aid in the planning for any needed concomitant treatment. For tibiofemoral lesions, the coronal plane alignment may be neutral, varus, or valgus and if the chondral lesion is in the compartment with increased physiologic load, realignment will need to be considered. A complete evaluation of the patellofemoral joint must include an assessment for malalignment as the initial results following autologous chondrocyte implantation of the patella or trochlea are not uniformly successful. With correction of malalignment via anteromedialization of the tibial tubercle, Minas et al[41] recently demonstrated similar results in the patellofemoral joint compared to the tibiofemoral joint. Similarly, meniscal and ligament deficiency must be corrected prior to or concomitant with articular cartilage restoration.

IMAGING

Standard anteroposterior standing radiographs allow assessment of the anterior tibiofemoral joint, but more commonly, tibiofemoral defects occur more posteriorly and are best assessed with a standing 45° flexion posteri-

or to anterior radiograph as popularized by Rosenberg et al.[48] Lateral radiographs demonstrate patellofemoral joint space and trochlear morphology and patellar tilt as described by Maldague and Malghem.[34] The patellofemoral joint is further assessed with an axial view in low degrees of knee flexion, such as the Merchant view. Clinical evidence of knee varus or valgus is documented with a hip-to-ankle view allowing measurement of the anatomic or mechanical axis, or both.

Although marked joint space narrowing on plain radiographs may help exclude the knee from cartilage restoration, MRI with two-dimensional fat suppression and three-dimensional fast-spin echo sequences can help delineate the focal lesion and, through volumetric studies, assess whether diffuse chondropenia is present. Moreover, MRI allows for evaluation of the subchondral bone for evidence of extensive edema, the extent and depth of osteochondritis dissecans, and the presence of avascular necrosis or fracture. Gadolinium enhancement may allow for assessment of proteoglycan content. Bone scan does not play a predictable role in evaluating articular cartilage, but may be useful in evaluating other pathologies such as bone tumor, stress reaction, and in some cases, complex regional pain syndrome. Independent of the findings on an imaging study, the final decision-making process is based on the nature and severity of the patient's symptoms and the defect-specific factors appreciated at arthroscopy.

ARTICULAR CARTILAGE LESION CLASSIFICATION

The International Cartilage Repair Society (ICRS) has devised a rational system to describe the depth of articular cartilage lesions; however, in the United States, most surgeons report articular cartilage lesions using a modified Outerbridge grading system.[45] Confusion can occur if the classifications are not explicitly stated. In the modified Outerbridge system, grade 3 represents deep fissuring of >50% depth with palpable but not exposed bone and grade 4 represents exposed bone without subchondral involvement. In contrast, the ICRS has four subcategories of grade 3 with 3c corresponding to the modified Outerbridge grade 4 (ie, exposed bone) whereas ICRS grade 4 represents bony involvement (Tables 1 and 2).

Independent of which classification system is used, it is important to document the defect characteristics and remain consistent. Along with the grade and depth, it is important to record the dimensions of the lesion using some type of intra-articular reference such as a sizing ruler to correct for magnification error or rarefaction due to the arthroscopic light. The position of the lesion may be recorded on a grid system as described by the Modified International Cartilage Repair Society Chondral Injury Classification System.[6,36] Further delineation of the local pathology character includes an assessment for bone loss or sclerotic change and the thickness of the surrounding cartilage (noting that the cartilage stiffness may be quantitated using commercially available probes). In addition, documentation of the degree of defect containment based on the condition of the surrounding walls allows planning for advanced restoration techniques where needed. As it may reflect on prognosis, an attempt is made to classify the lesion as acute or chronic.

TABLE 1
MODIFIED INTERNATIONAL CARTILAGE REPAIR SOCIETY (ICRS) GRADING SYSTEM

Injury Grade	Modified ICRS
0	Normal cartilage
1a	Soft indentation
1b	Superficial fissures and cracks
2	Lesions extending down to <50% of cartilage depth
3a	Defects extending down >50% of cartilage layer
3b	Defects down to calcified layer
3c	Defects down to but not through the subchondral boneayer
3d	Delamination
4	Severely abnormal; with penetration through subchondral plate

KNEE CLASSIFICATION

As noted previously, many articular cartilage lesions are associated with other knee pathologies including tibiofemoral varus/valgus malalignment, patellofemoral malalignment as described by Fulkerson,[20] ligament insufficiency, and meniscal deficiency. Bipolar change or asymptomatic disease in other knee compartments may also factor into the decision-making process. A comprehensive strategy must be developed to manage these concomitant pathologies with respect to the timing of treatment.

PATIENT CLASSIFICATION

Patient-specific factors play a significant role in the decision-making process. Baseline information should include chronologic and biologic age, body mass index, current and desired activity level, and comorbidities that could potentially impede a successful result (ie, inflammatory disease, fibromyalgia, other involved joints). An understanding of the impact that the patient's condition has on his or her general quality of life is also critical to establish, as this factor may become the primary outcome variable to measure a successful result. Finally, it is

TABLE 2
OUTERBRIDGE GRADING SYSTEM

Injury Grade	Outerbridge Classification	
	Original	Modified
I	Softening and swelling of cartilage	Softening and swelling of cartilage
II	Fissures and fragmentation in an area <1/2 inch in diameter	Fibrillation/fissuring <50% of cartilage depth (report dimensions separately)
III	Fissuring and fragmentation in an area with >1/2 diameter involvement	Fibrillation/fissuring >50% cartilage depth without exposed bone
IV	Erosion of cartilage down to exposed but not penetrated subchondral bone	Erosion of cartilage down to exposed but not penetrated subchondral bone

important to understand the patient's willingness and ability to undergo the rigors of the requisite postoperative rehabilitation and the time required to achieve a successful result.

SURGICAL DECISION MAKING

In light of the extended time for postoperative recovery, the resource intensity, and the variability in treatment efficacy, it is critical that the patient receive the treatment that optimally matches the articular cartilage lesion, the overall condition of the knee, his or her expectations, and lifestyle; in other words, a "demand-match approach."[34] The aggregate demand of these factors is in essence matched to the available treatment option. Due to the expanding armamentarium and knowledge base of cartilage restoration treatments, it is important not only to review new technologies, but also search for well-designed prospective studies of established treatments that could change their place in the demand-match algorithm. A few comments with respect to demand matching are appropriate for each technique.

Nonoperative

This is often used for articular cartilage lesions of two extremes: the small (<1 cm^2) asymptomatic lesion and extensive lesions that are outside the realm of standard current cartilage restoration techniques, typically described as degenerative arthritis. For the smaller lesions, nonoperative treatment includes observation and patient education regarding what symptoms are characteristically associated with defect progression. In the near future, additional parameters such as volumetric MRI or cartilage-specific imaging might influence the decision to surgically intervene to maximize the benefit of a specific treatment option. Classic nonoperative options include physical therapy to optimize muscle strength and balance, unloader bracing, disease modifying agents (eg, glucosamine/chondroitin sulfate), non-narcotic analgesics, viscosupplementation, and pulsed low amplitude electrical stimulation.[20]

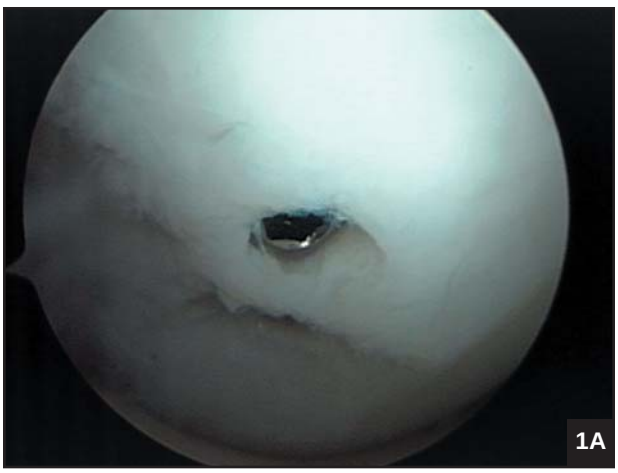
Palliative

Arthroscopic debridement and lavage is considered only as a palliative first-line treatment for articular damage and for treatment of the incidental or unsuspected small cartilage defect (ie, <2 cm^2). Simple irrigation to remove debris may temporarily improve symptoms in up to 70%, and when combined with chondroplasty, the success rate may initially increase.[3,14,29,30,37,46,50,55] Despite a well-popularized, randomized study of arthritis treated with arthroscopic debridement showing no improvement over sham surgery,[43] this should not reflect on the use of arthroscopic stabilization chondroplasty for articular cartilage lesions that are associated with mechanical symptoms or used with the objective of reducing the effects of biologically active debris leading to synovial irritation and effusion. Clearly, however, arthroscopic debridement is unlikely to lead to long-lasting relief as supported by the literature.[3,14,29,30,37,46,50,54]

Reparative

Although articular cartilage has minimal ability to heal without intervention, scenarios exist in which implementing a "reparative" option is appropriate. Osteochondral fractures and osteochondritis dissecans represent the obvious clinical problems where primary repair is likely to be implemented. Obviously, it is the bone that is repaired; marginal integration of the cartilage often does not occur. Clearly, the best opportunity to achieve a predictably good or excellent result is when successful osteochondral healing is achieved. As the attached articular cartilage remains alive in synovial fluid, the goal is to promote bone-to-bone healing with restoration of a congruent joint surface using basic biologic and mechanical principles implemented in articular fracture care (establishing an appropriate healing response with debridement, bone grafting where needed and rigid fixation with compression) (Figure 1).

The other procedures that may be termed reparative in nature are grouped together as marrow stimulation techniques. These are cost-effective and easily implemented at the time of lesion diagnosis. Several types of treatments



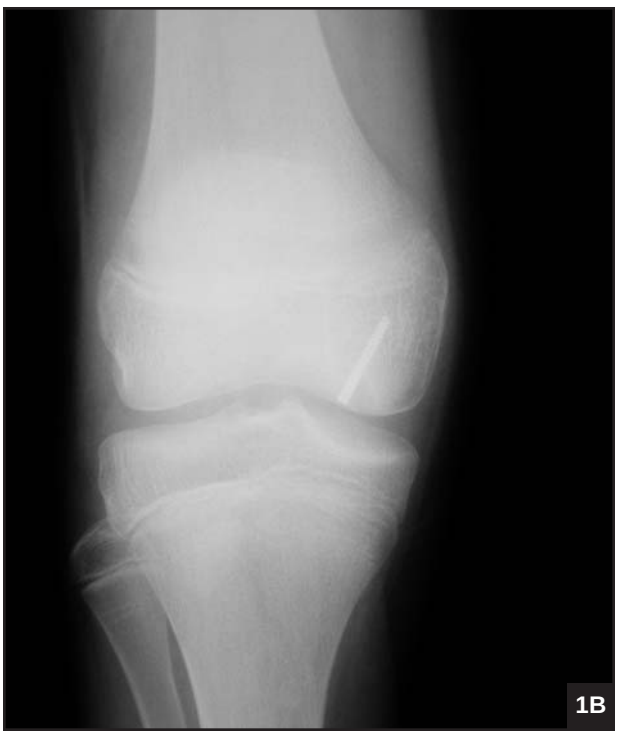


Figure 1. The goal for successful osteochondral healing is to promote bone-to-bone healing with restoration of a congruent joint surface using basic biologic and mechanical principles implemented in articular fracture care. Arthroscopic photograph of an anatomically reduced lesion of osteochondritis dissecans treated with a cannulated compression screw (A). Plain radiograph demonstrating the lesion and screw with the head of the screw at the subchondral bone (B).

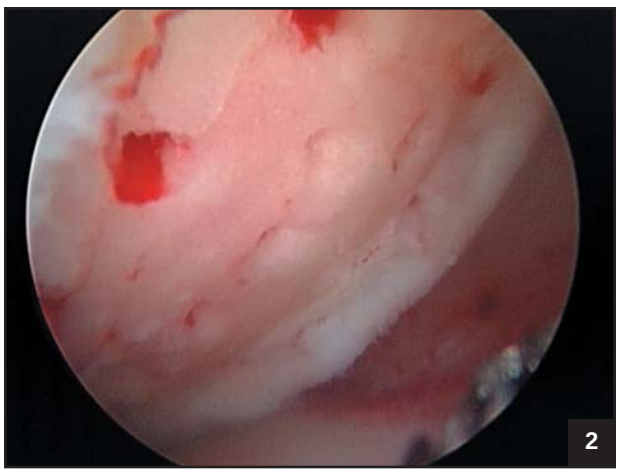
use this technique including microfracture, subchondral drilling, and abrasion arthroplasty. These procedures are recommended in active patients with moderate symptoms that have smaller lesions (<2 cm^2) or in lower-demand patients with larger lesions (>2 cm^2). Microfracture is the preferred marrow stimulation technique because it creates less thermal energy compared to drilling and provides a controlled depth of penetration with holes made perpendicular to the subchondral plate, which otherwise remains intact.

A successful result is predicated on appropriate surgical technique, which includes creating a vertical wall in the surrounding normal cartilage at the transition zone, careful violation of the calcified layer at the base of the defect, and penetration of the subchondral bed with small holes spaced 2-3 mm apart (Figure 2).[19] The outcome appears to be dependent on strict patient compliance with continuous passive motion or manual repetitive cycles and nonweight bearing for approximately 6 weeks for femoral condyle lesions or weight bearing in extension with limited flexion for patellofemoral lesions. Reports vary as to the efficacy of marrow stimulation techniques. Positive outcomes, though, appear to be related somewhat to smaller monopolar defects in patients whose activity and demand levels do not exceed the ability of fibrocartilage repair tissue to lead to symptom reduction. Results in appropriately selected patients exceed 80% good and excellent with a high return to sport in the intermediate term with appropriately indicated patients.[5,22,39,51-53]

Restorative

Restorative options include osteochondral grafts and autologous chondrocyte implantation. Osteochondral grafts restore the articular surface by implanting a cylindrical plug of subchondral bone and articular cartilage. The source of the tissue can be from the host (autograft) or from a cadaveric donor (allograft). Several challenges face autograft and allograft transplants: edge/marginal integration, restoring three-dimensional surface contour, and graft availability.

Osteochondral autografts are advantageous by virtue of using the patient's own tissue, eliminating immunological concerns and the concerns of cell attrition with use of cultured/refrigerated allografts. This technique is limited by the size of the graft (<1 cm^2) and involves obtaining the donor osteochondral graft from a nonweight bearing area of the joint and placing it into the prepared defect site (Figure 3). The major risk involved with osteochondral autografts is plug failure and donor-site morbidity, which increases as the size of the harvested plug increases. Postoperative rehabilitation includes early range of motion and nonweight bearing for 2 weeks with an increase to full-weight bearing from 2-6 weeks. Indications for use of this technique include primary treatment of smaller lesions considered symptomatic in relatively high-demand patients and for similarly sized lesions for which a microfracture or possibly prior autologous chondrocyte implantation procedure failed. Following these indications, the results have been favor-



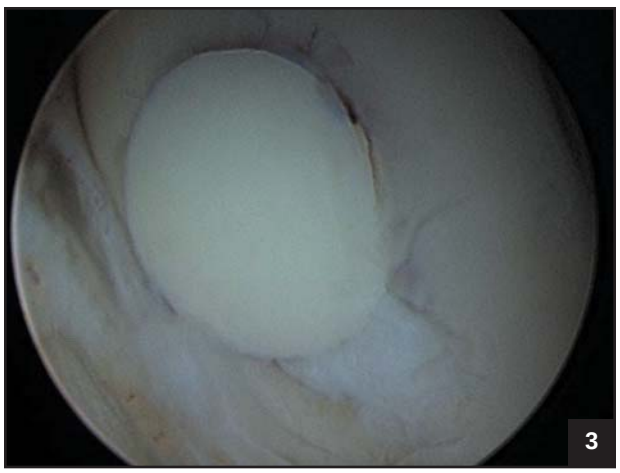
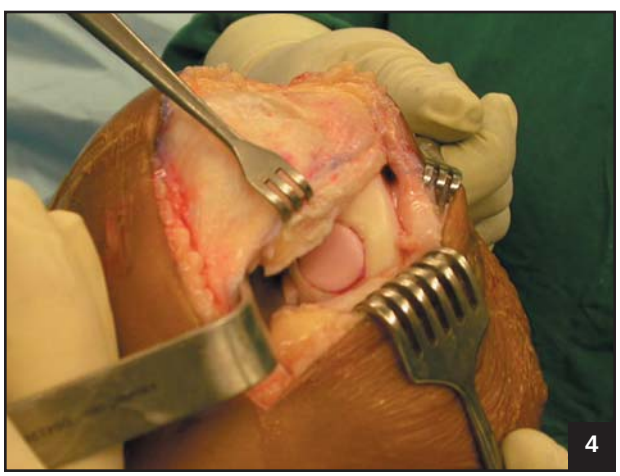


Figure 2. Arthroscopic photograph of a 2 cm^2 lesion treated with microfracture. Note the creation of vertical walls with careful violation of the calcified layer at the base of the defect and penetration of the subchondral bed with small holes spaced 2-3 mm apart. **Figure 3.** Arthroscopic photograph of a lesion previously treated with microfracture resulting in adequate fibrocartilage fill with persistent symptoms revised with a 10-mm osteochondral autograft plug. **Figure 4.** Intraoperative photograph of a lesion of osteochondritis dissecans treated with a fresh osteochondral allograft.



able with intermediate-term follow-up.[25-27,31,32]

Osteochondral allografts are used to treat larger defects (>2 cm^2), which are difficult to treat with other methods. Osteochondral allografts involve the transplantation of mature, normal hyaline cartilage with intact native architecture and a percentage of viable chondrocytes. Because the graft includes subchondral bone, any disorder with associated bone loss (eg, avascular necrosis, osteochondral fracture, and osteochondritis dissecans) lends itself to osteochondral treatment (Figure 4). Tissue matching and immunologic suppression are unnecessary as the allograft tissue is relatively immunoprivileged as the bone portions are devitalized and the chondrocytes are sequestered in the matrix. Graft preservation techniques include fresh, frozen, and prolonged cold preserved. Fresh allografts must be used within 3-5 days of procurement and thus logistic concerns become an issue, furthermore, with the current regulatory environment in the United States these truly fresh grafts are not available for implantation. Frozen grafts can be stored and shipped on demand potentially alleviating scheduling issues; however, frozen osteochondral tissue lacks cellular viability. The prolonged cold preservation method increases the "shelf life" of the graft possibly to as long as 28 days and alleviates the scheduling difficulties while maintaining cell viability (78% at 28 days preservation); however, chondrocyte viability remains an issue after implantation.[1,12,33] Postoperative rehabilitation consists of immediate continuous passive motion and protected weight bearing for 6-8 weeks. This procedure is most often used as a secondary treatment option in patients who have failed previous attempts at cartilage repair or high-demand patients with large lesions, especially those that involve the subchondral bone. The results have been some of the most favorable, with the longest follow-up to date in the literature for cartilage restoration options.[2,5,8,9,11,21,24] Extreme caution must be exercised when extrapolating the past success from acute allograft transplantation to the current use of refrigerated specimens. Furthermore, the above discussion pertains to discrete segmental allografts (Mega OATS) and not to the large osteochondral shells or bipolar transplantations.

Autologous chondrocyte implantation is a two-stage procedure involving a biopsy of normal articular cartilage (300-500 mg), usually obtained through an arthroscopic procedure, in which the cartilage is harvested from a

minor load-bearing area (upper medial femoral condyle or intercondylar notch). These chondrocytes are then cultured in vitro and implanted into the chondral defect beneath a periosteal patch during a second-stage procedure that requires an arthrotomy (Figure 5). This restorative procedure results in "hyaline-like" cartilage, which is believed to be biomechanically superior to fibrocartilage.[23] Postoperative rehabilitation entails continuous passive motion and protected weight bearing for up to 6 weeks.

Autologous chondrocyte implantation is most often used as a secondary procedure for the treatment of medium to large focal chondral defects (>2 cm^2). Considering the cost of the procedure and the need for two surgeries, it is typically not used for smaller lesions (<2 cm^2) that can be managed with a single surgery and with much less expense (eg, osteochondral autograft and marrow stimulation). For the larger lesions, in light of some concern for the long-term durability of prolonged-fresh refrigerated allografts and the need to breach the subchondral bone, autologous chondrocyte implantation is typically used for the younger patient with relatively high physical demands. Results in appropriately indicated patients are $\geqslant$85% good and excellent. These results must be tempered by the report of Micheli et al[38] who reported superior results with autologous cultured chondrocyte implantation when the procedure is performed early after the onset of cartilage pathology and poor results with delayed treatment. These studies suggest that the remaining articular cartilage is deleteriously affected during periods of abnormal loading over time.[4,7,13,28,40,44,47,55]

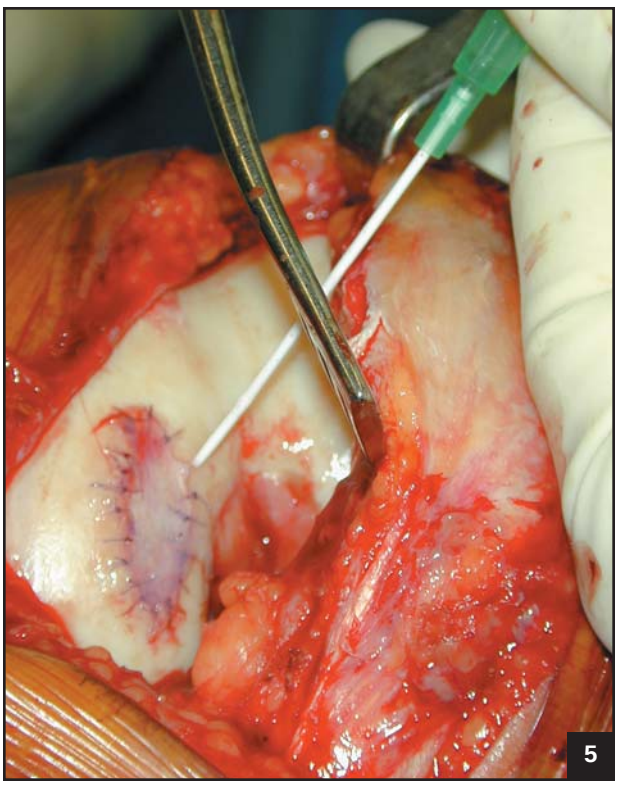


Figure 5. Intraoperative photograph of a chondral lesion treated with autologous chondrocyte implantation.

CONCLUSION

The applications of the different treatment options for articular cartilage defects overlap. Nonetheless, it is important for the cartilage surgeon to be skilled in all techniques and thus not limit the patient to only a single treatment the surgeon champions, ie, one technique does not optimally treat all articular cartilage lesions for all patients. Even more important is the focus on appropriate decision making rather than on the details of any single surgical technique. When developing one's own demand-match treatment algorithm, six factors are extremely important noting that one of these factors is relatively important especially in societal terms—resource intensity and expense. The five other factors are 1) the lesion classification, including size; 2) knee factors, including alignment, meniscal and ligamentous status; 3) patients factors, including activity demand and relative age; 4) whether the treatment is primary or secondary (treating a failed primary treatment); and 5) the genetically guided baseline of the patient's articular cartilage quality (Appendix). In general, the older patient with lower demands with low level symptoms is served best with a cost-efficient primary treatment including debridement, osteochondral autograft, and marrow stimulation whereas the younger patient with intermediate-sized lesions may be initially managed with osteochondral autograft or marrow stimulation with close observation and those with larger lesions may be treated with either marrow stimulation or debridement in anticipation of autologous chondrocyte implantation should these measures fail. Although we provide an overview treatment algorithm, it will need updating on a regular basis as outcome data are reported for current treatments and new treatments that will be clinically available (Figure 6).

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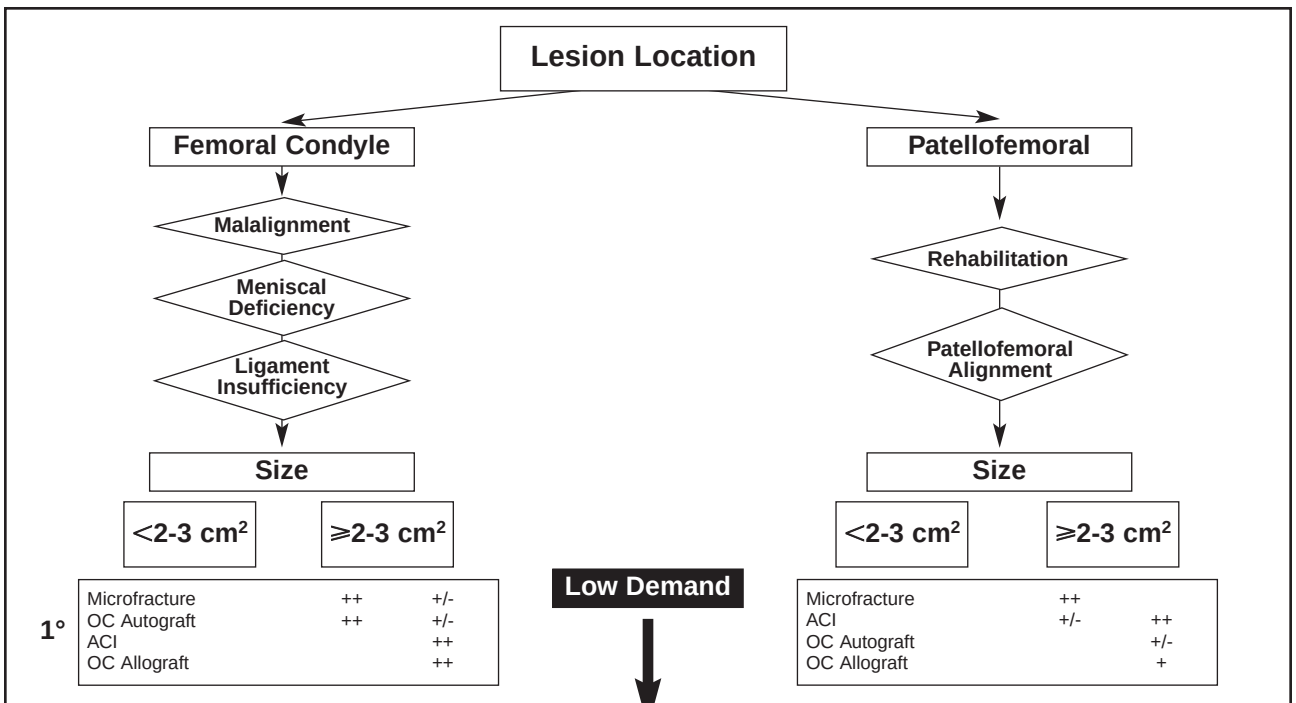

Figure 6. Overview treatment algorithm for articular cartilage defects. Abbreviations: ACI=autologous chondrocyte implantation and OC=osteochondral.

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APPENDIX ONE

Genetic Implications on Articular Cartilage Quality

Appreciating the complexity of the cellular and extra cellular matrix make up of articular cartilage, it comes with no surprise that the number of genes controlling or influencing this composite is enormous. With the aid of more focused genetic assessment using microarrays, it is becoming increasingly evident that not all articular cartilage is created equal. Granted, degenerative joint disease is multifactorial, but genetic influences have been attributed to up to 30% for knee osteoarthritis. Although not directed or indicated for the treatment of osteoarthritis, it follows that a segment of cartilage restoration patients will indeed having underlying genetic factors that place them in the time continuum of osteoarthritis. In other words, although two young patients with osteochondritis dissecans may not have evidence of osteoarthritis and appear to be in a common category, their underlying genetics may influence their near and long-term outcomes. Although the ability to genetically classify a patient's articular cartilage is currently not available as an established clinical tool, it is worthwhile to discuss this pending ability and its implications for treatment. Genetic defects will involve a multitude of cellular and matrix pathologies with extensive overlap, but to gain an appreciation we will assume a simplistic grading scale as follows:

ARTICULAR CARTILAGE (AC) QUALITY GRADING

Grade	Quality	Severity of Genetic AC Defects
A	Normal	None
B	Nearly normal	Minor
C	Abnormal	Moderate
F	Severely abnormal	Extensive

In this crude hypothetical grading system, the clinical relevance of the two extremes are well recognized: the 39-year-old patient without other risk factors for osteoarthritis presents with joint space loss or the 85-year-old patient with pristine articular cartilage. As one applies the various options of cartilage restoration, keeping the quality of the "noninvolved" articular cartilage in mind will potentially be an additional factor in demand-match optimization of treatment.

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