Review Article

Clinical Evaluation and Preoperative Planning of Articular Cartilage Lesions of the Knee

Nathan A. Mall, MD
Joshua D. Harris, MD
Brian J. Cole, MD, MBA

Abstract

Articular cartilage injuries are quite common. Most studies and review articles on cartilage repair and restoration focus on the different techniques available to treat cartilage defects; however, few thoroughly discuss the initial evaluation of patients with these defects. Outcomes are intimately associated with appropriate patient selection and indications for treatment; therefore, understanding the initial evaluation and conservative treatment of cartilage defects is essential to achieving excellent outcomes after surgical intervention, regardless of the chosen procedure. In patients with cartilage injury, a careful history, physical examination, and imaging are required before treating the lesion to ensure the patient's symptoms are actually related to the defect. To address any special considerations, other factors must be considered to improve patient outcomes, including the status of the meniscus, assessing and treating malalignment or offloading the patellofemoral compartment, and reconstructing any ligamentous deficiencies. It is important for medical providers to understand what cartilage lesions may be symptomatic and when to refer these patients to surgeons who manage cartilage injury.

From Regeneration Orthopedics, St. Louis, MO (Dr. Mall), Houston Methodist Hospital, Houston, TX (Dr. Harris), and the Center for Cartilage Restoration, Rush University Medical Center, Chicago, IL (Dr. Cole).

J Am Acad Orthop Surg 2015;23: 633-640

http://dx.doi.org/10.5435/JAAOS-D-14-00241

Injury to articular cartilage is likely more common than originally thought because newer imaging techniques now identify damage to cartilage matrix following anterior cruciate ligament (ACL) injury, despite the normal appearance of the cartilage at the time of arthroscopy.[1] This finding indicates that any type of impaction injury or significant increase in force, whether the result of malalignment or meniscal deficiency, may cause injury to the cartilage ultrastructure or chondrocytes that later leads to macroscopic cartilage damage. The impact of subchondral bone edema, as seen on MRI, also has been recognized as a significant contributor to pain and symptoms from cartilage injury.[2] With these defects, the evaluation process is complicated because many cartilage injuries are asymptomatic and the natural history of these defects is not fully understood.[3] However, most authors would agree that symptomatic defects are likely to remain symptomatic and possibly worsen over time,[4,5] which may also correlate with an increase in the size of the defect.[6] Symptomatic, focal, International Cartilage Repair Society grade III or IV cartilage defects can be treated with cartilage restoration procedures. New cartilage restoration techniques continue to be developed, with randomized controlled trials demonstrating promising results;[7,8] however, unless appropriate clinical indications are understood and preoperative evaluation is performed, any cartilage restoration technique is likely to fail.

Clinical Presentation

The true incidence of full-thickness articular cartilage lesions in the knee has not been well studied, but it is thought to be between 5% and 10%.[3,9] However, this estimate comes from arthroscopic studies and the true number could be higher when considering the number of asymptomatic cartilage defects. A systematic review by Flanigan et al[10] found a prevalence of full-thickness cartilage defects in 36% of 931 athletes across 11 studies. Of these defects, 14% were asymptomatic; in basketball players, this number increased to 59%. Most patients with articular cartilage lesions that require treatment report pain, typically with weight bearing, and localized to the same compartment as the defect.[6] Some patients have little pain but report swelling of the knee with activity. Patellofemoral cartilage lesions typically exhibit a similar pain profile and pattern as patellofemoral pain syndrome. Patellofemoral pain syndrome can sometimes be differentiated from articular cartilage pain by the presence or absence of swelling because patellofemoral pain syndrome typically does not cause the knee to swell.[6] Patients with femoral condyle and tibial plateau lesions typically present with joint line tenderness and may have mechanical symptoms. Pain may be worsened by weight bearing, or, for more posterior lesions, with the knee in flexion. Because pediatric and adolescent patients may not be able to accurately localize their pain, the presence of a limp or swelling may indicate a symptomatic defect.

On average, patients with articular cartilage defects who are considering cartilage restoration treatments have previously undergone more than two procedures.[11] In these situations, obtaining previous intraoperative photographs or directly communicating with the previous surgeon may obviate the need for a diagnostic planning arthroscopy. Under-rehabilitation following past surgeries is a common source of symptoms in this patient population and must be ruled out as a pain generator before proceeding with cartilage restoration surgery.

Physical Examination

Because cartilage defects can be asymptomatic, it is essential to confirm physical examination findings that are consistent with a cartilage injury before performing cartilage restoration. The examination should also focus on detecting potential contraindications to cartilage restoration and determining if concomitant procedures will be required to optimize results.[12] Standing and gait analysis should be performed to evaluate for malalignment or specific gait abnormalities. Rotational deformity in the lower extremity and muscular imbalance have also been shown to alter the forces and biomechanics of the knee joint.[13] Therefore, a thorough examination should include assessment of hip rotation, hamstring and quadriceps strength and flexibility, and foot alignment. Muscle weakness, which can contribute to knee pain, can be assessed by manual muscle strength testing or via instrumented strength testing. Because ligament insufficiency has been shown to adversely affect cartilage restoration,[14] it is essential to thoroughly examine the ACL, posterior cruciate ligament (PCL), collateral ligaments, and the posterolateral and posteromedial corners. To rule out any rotational instability, a pivot shift examination should be performed both in the office and before any surgical procedure, especially when a prior ACL reconstruction is placed in a slightly vertical position.

Joint line tenderness may be present in varying degrees of flexion, depending on the location of the cartilage defect, with pain typically worse directly over the cartilage defect. The Wilson test can be performed for diagnosis of an osteochondral defect. Kocher et al[15] found that the sensitivity of the clinical examination for the diagnosis of osteochondritis dissecans was 90.9% and the specificity was 69%, which was greater than MRI.

Imaging

Radiography

An AP extension weight-bearing view and a PA 45° flexion Rosenberg view should be obtained to assess for tibiofemoral joint space narrowing. Lateral and sunrise or Mercer-Merchant views can be used to assess the patellofemoral articulation.

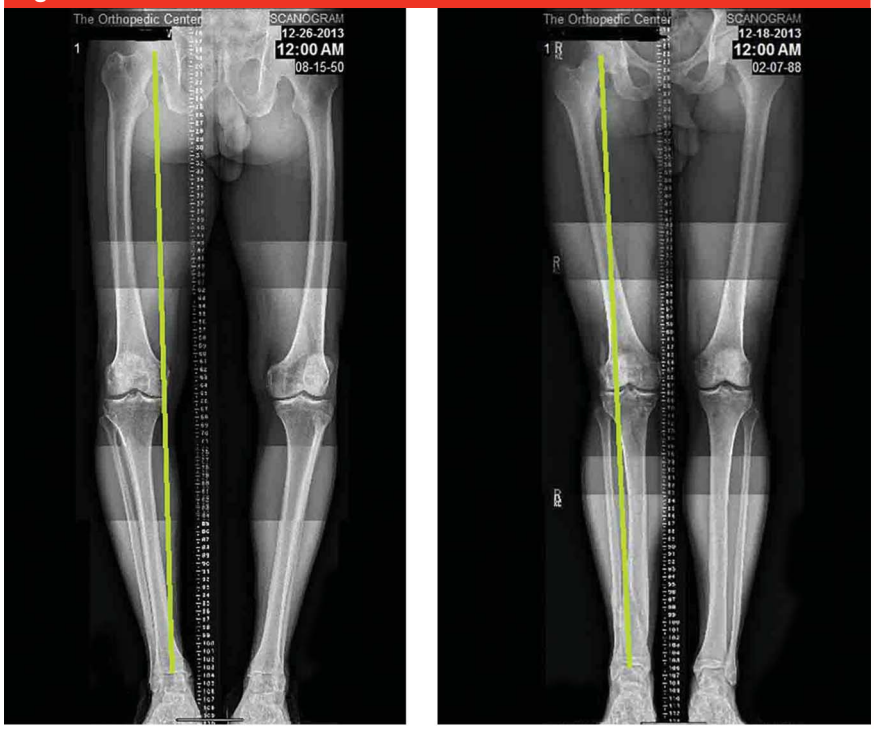
Dr. Mall or an immediate family member is a member of a speakers' bureau or has made paid presentations on behalf of Arthrex. Dr. Cole or an immediate family member has received royalties from Arthrex and DJO Global; serves as a paid consultant to Arthrex, Carticept, Regentis, and Zimmer; has stock or stock options held in Carticept and Regentis; has received research or institutional support from Arthrex, Medipost, the National Institutes of Health (National Institute of Arthritis and Musculoskeletal and Skin Diseases and National Institute of Child Health and Human Development), and Zimmer; has received nonincome support (such as equipment or services), commercially derived honoraria, or other non-research–related funding (such as paid travel) from Athletico, Össur, Smith & Nephew, and Tornier; and serves as a board member, owner, officer, or committee member of the American Academy of Orthopaedic Surgeons, the American Orthopaedic Society for Sports Medicine, the American Shoulder and Elbow Surgeons, the Arthroscopy Association of North America, and the International Cartilage Repair Society. Neither Dr. Harris nor any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article.

We typically obtain our standard knee series with a sizing marker to correct for magnification, which can be used for sizing should meniscal or osteochondral allograft procedures be performed. Standing full-length alignment radiographs should be obtained as part of the assessment of patients undergoing cartilage restoration procedures (Figure 1). Measurements of the anatomic and mechanical axes are drawn to assess for malalignment.

MRI

MRI is indicated in patients who have signs and symptoms consistent with intra-articular pathology (ie, pain, swelling, or mechanical symptoms), despite having relatively normal weight-bearing radiographs. Once significant joint space narrowing has occurred, the extent of cartilage and meniscal damage is likely too great to allow for cartilage restoration procedures. Gradient echo sequences are often used as cartilage-specific images because of their ability to differentiate articular cartilage from the surrounding joint fluid and subchondral bone. However, these sequences do not adequately show internal signal within the articular cartilage or subchondral edema. Therefore, a T2-weighted and/or short tau inversion recovery fluid-sensitive series should be used to assess for these important factors. Newer MRI techniques, such as T1rho mapping, T2-mapping, sodium imaging, and delayed gadolinium-enhanced MRI of cartilage[16] were initially implemented in the research setting; however, these techniques are becoming more common in the clinical setting, allowing for the investigation of articular cartilage ultrastructure and morphology as well as cartilage biology and metabolism[16] (Figure 2).

Figure 1



A B

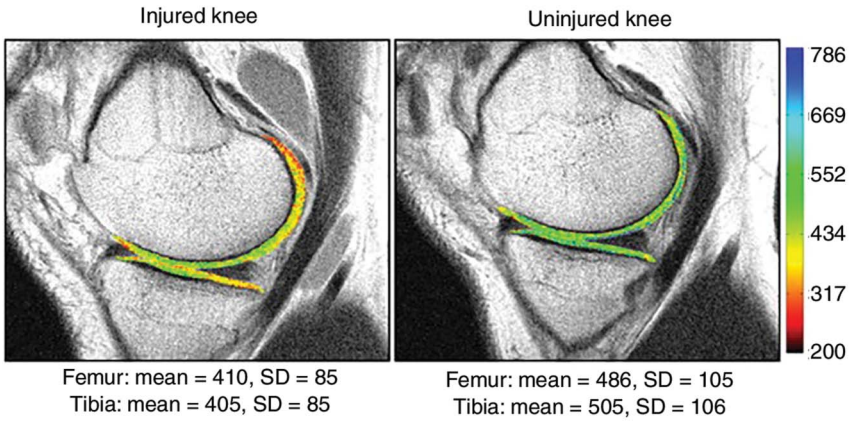
Full-length standing radiographs demonstrating varus (**A**) and valgus (**B**) malalignment. When cartilage restoration procedures are indicated, malalignment should be addressed either at the same time as the cartilage restoration procedure or in a staged manner to improve outcomes.

Special Considerations

Ligament Status

Careful examination and objective testing of the knee ligaments must be performed before cartilage restoration procedures. In a controlled laboratory ex-vivo study to simulate cartilage restoration procedures, Efe et al[14] found that ACL deficiency was more likely to cause disruption of the gel material placed into medial femoral condyle defects. Therefore, the ACL must be functional and prevent not only anterior translation of the tibia but also rotational forces to protect the cartilage restoration. Vertical ACL grafts can continue to allow rotational forces similar to an ACL-deficient knee. Careful pivot shift examination must be performed intraoperatively, and if concern for rotational instability is present, concomitant ACL revision must be considered at the time of cartilage restoration procedures. PCL injury has been shown to cause a predisposition to medial femoral condyle and patellofemoral cartilage lesions. One study found that nearly 80% of chronic PCL injuries had medial femoral condyle defects and 50% had patellofemoral defects.[17] In a study of multiligamentous and posterolateral corner reconstructions, 14 of 21 patients had some cartilage injury at the time of surgery; those patients had worse outcomes at a mean 87-month follow-up.[18] Thus, ligamentous deficiency and cartilage injury have an intimate relationship, with ligament injury causing cartilage defects and cartilage defects potentially affecting

Figure 2



T1rho maps of the medial compartment of the tibia and the femur that were generated using the MRIMapper software package (Beth Israel Deaconess Medical Center). The mean delayed gadolinium-enhanced MRI of cartilage indices were calculated. The blue and red regions denote high and low glycosaminoglycan concentrations, respectively. (Reproduced with permission from Fleming BC, Oksendahl HL, Mehan WA, et al: Delayed gadolinium-enhanced MR imaging of cartilage dGEMRIC following ACL injury. *Osteoarthritis Cartilage* 2010;18:662-667.)

long-term outcomes after ligament injury and reconstruction.

Meniscus Status

The meniscus has multiple functions in the knee, one of which is to convert forces across the tibiofemoral joint into radial-directed hoop stresses. This allows the meniscus to transmit 50% of the joint load when the knee is extended and 90% when the knee is flexed.[19] Loss of these hoop stresses, such as in a radial tear necessitating a wedge resection of as little as 20% of the meniscus, can result in contact forces >350% of normal.[20] Loss of the meniscus leads to point loading that can dramatically change the forces seen in the cartilage and subchondral bone and lead to significant injury. Verma et al[21] found that the contact area decreased by an average of 46% following medial meniscectomy; therefore, restoration of cartilage should not be performed without the protection of a competent meniscus, either via repair or allograft transplantation.

Other roles for the meniscus include increasing the surface area of contact and the congruency of the joint as well as assisting in synovial fluid mechanics. Friction in a knee with a partial or complete meniscectomy is increased by >20%.[22] Articular cartilage obtains its nutrition from synovial fluid; thus, without a meniscus the normal fluid extrusion and circulation that occur with meniscus compression is lost. The posterior horn of the medial meniscus is also a secondary stabilizer of the anterior-to-posterior forces in the knee, becoming important in the setting of ACL deficiency or following ACL reconstruction.

Meniscus transplantation can restore forces to more normal levels and distributions.[21] Following a medial meniscus transplant, the contact area was not statistically different from the intact state.[21] Because allograft meniscal transplantation can also restore many of the other important roles of the meniscus, it should be considered in combination with other cartilage restorative procedures, either concomitantly or in a staged fashion. Inferior results for cartilage restoration can be expected in the setting of meniscal-deficient knees when meniscal deficiency is not addressed.

Body Mass

Increased body mass has been shown to lead to increased forces on the weight-bearing joints. One study evaluating the results of microfracture demonstrated that poorer results were associated with a body mass index (BMI) >30.[23] A recent systematic review demonstrated that BMI does not solely affect the International Knee Documentation Committee subjective score (validated for patients with articular cartilage injury) following autologous chondrocyte implantation (ACI).[24] Nevertheless, the proliferating cells that begin to form a cartilage matrix in the early postoperative setting of an ACI procedure could very well be affected similarly to cells following a microfracture. However, fresh osteochondral allograft tissue with intact cartilage matrix and living chondrocytes may be less affected by an increased body mass, especially if any malalignment or meniscal deficiency producing excess forces or point loading was addressed at the time of implantation.

Bone Edema

Recent literature has led to the consideration of the cartilage and bone as one osteochondral unit rather than individual entities.[25] Alterations in the osteochondral unit occur in chronic cartilage defects and in those previously treated with marrow stimulation techniques, such as microfracture, drilling, or abrasion arthroplasty.[2,23] These changes include thickening and stiffening of the subchondral plate that causes thinning of the articular cartilage, which may leave the cartilage more susceptible to shear forces.[26] When bone edema is present, we typically recommend replacing both

the cartilage and the bone, using either an osteochondral autograft or allograft transplant, depending on the size of the lesion (Figure 3). Subchondral calcium phosphate may also be used for symptomatic bony lesions.[27] This can be done in conjunction with cartilage restorative procedures, such as ACI; however, the results of such a procedure have not been proven. Other techniques include the sandwich technique used with ACI. This technique requires bone grafting, which is then sealed with periosteum or collagen bilayer membrane and fibrin glue. Another membrane is sewn into the cartilage per standard ACI technique and the chondrocytes are injected between the two periosteal or collagen membrane layers.[28]

Malalignment

Malalignment significantly increases forces in the affected compartment. Standing radiographs are used to determine if malalignment exists and to measure the correction needed. Alignment is typically corrected to a point that is 62% across the medial-to-lateral length of the tibial plateau in the setting of osteoarthritis; the goal is to markedly offload the diseased compartment.[29] However, in the setting of cartilage restoration, we typically choose a point that is midline or just slightly overcorrected (ie, the opposite tibial spine) as the correction goal to redistribute the forces so that the regenerated or transplanted cartilage experiences normal forces but does not overload the opposite compartment.

Similar to the tibiofemoral joint, excess forces can mitigate results of cartilage restoration in the patellofemoral joint. Outcomes of patellofemoral cartilage restoration with unloading osteotomy are significantly better than outcomes of isolated patellofemoral cartilage restoration without osteotomy[30] (Figure 4). Some authors believe that every patellar or trochlear lesion should be offloaded at the time of cartilage restoration to improve the success of the procedure.[31,32] This is typically performed using a tibial tubercle osteotomy in which the tubercle is moved anterior and medial. In patients with proximal-medial patellar defects, there is some concern that the pressure may increase in this location with medialization of the tubercle;[33] therefore, straight anteriorization is recommended.[32] Isolated trochlear defects may not require an osteotomy, depending on the location; however, this question has not been answered in the literature and further studies need to be performed.

Defect Size

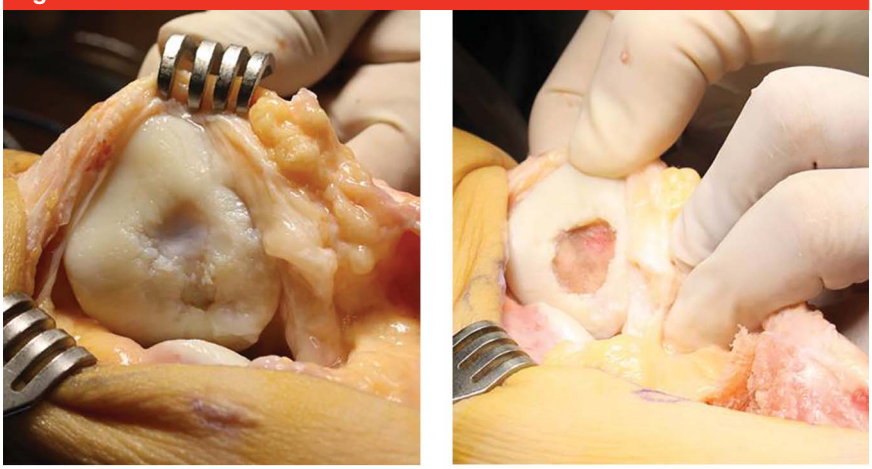
The size of the lesion is one of the major determining factors when deciding what type of procedure should be performed. However, lesion size may also be a factor in whether or not a defect is or becomes symptomatic. A study by Guettler et al[34] demonstrated that a lesion >1 cm in diameter was increasingly associated with symptoms. However, the true association with size is likely related in part to the ratio of lesion size to the overall size of the femoral condyle. A 1-cm defect is likely to be much more clinically relevant when it takes up 50% of the width of the condyle compared with a very large condyle in which a 1-cm defect may make up only 20% of the width. Therefore, a 2-cm^2 lesion of the medial femoral condyle may be more likely to produce symptoms in a 100-lb, 4′10″ female gymnast versus a 300-lb, 6′7″ male football player.

Smaller lesions allow for more options for cartilage restoration. Small, isolated cartilage defects may be treated with microfracture, microfracture plus a scaffold, ACI, minced allograft articular cartilage, osteochondral autograft, or osteochondral allograft procedures. In defects affecting the bone and cartilage, osteochondral autograft and allograft can be used. Lesions >2- to 4-cm^2 are typically treated with ACI, minced allograft articular cartilage, or osteochondral autograft. Osteochondral autograft is limited by the availability of non–weight-bearing cartilage in the knee and donor site morbidity. Studies evaluating the outcomes after microfracture have found that larger lesions were correlated with worse outcomes and that larger defects may have poor defect fill,[23,35] which also correlated with inferior outcomes.[23] Whereas microfracture using an adjunct scaffold of freeze-dried allogeneic articular cartilage combined with platelet-rich plasma may improve the ability to use this technique to manage a larger surface area defect, clinical results are forthcoming.[36] Other scaffolds[8,37] and cartilage treatment options[38,39] are currently being studied, with few available clinical results; however, the discussion of cartilage restoration techniques is beyond the scope of this article.

Figure 3

Magnetic resonance image demonstrating bone edema that can be present with a cartilage injury. The patient has cartilage defects of the medial femoral condyle and the lateral tibial plateau.

Figure 4



A B

Intraoperative clinical photographs demonstrating a typical cartilage defect of the patella before débridement (**A**) and after débridement (**B**). Also, a tibial tubercle osteotomy was performed. We typically recommend performing the osteotomy first, with fixation delayed until after the cartilage restoration procedure of choice has been performed. We do not routinely flip the osteotomy up to gain access to patellar or trochlear defects. The osteotomy itself adds some added mobility to the patella, and we prefer to leave the inferior cortex intact, creating a greenstick fracture.

Defect Location

Lesions located on the femoral condyles allow for the greatest span of potential cartilage restoration techniques. Depending on the size of the lesion, microfracture, microfracture plus a scaffold, ACI, minced allograft articular cartilage, or osteochondral transplantation can be used. The treating surgeon must carefully identify and address any injury to the subchondral bone. Osteochondral transplants are ideal for articular cartilage lesions located on the femoral condyle, especially when the underlying subchondral bone is involved. Larger defects or defects affecting the bone on the patella or the trochlea are more difficult to treat. Osteochondral allografts are size-matched, not shape-matched. The variable topography of the trochlea and patella is difficult to match in these larger defects; therefore, the use of ACI, minced cartilage, or microfracture plus a scaffold is much more desirable for lesions of the patella and trochlea.

Because the tibial plateau cartilage is much thinner than that of the femoral condyle or the cartilage of the patellofemoral joint, it is more difficult to treat and available cartilage restoration techniques are much more limited. When tibial plateau defects are encountered at lower grades of injury (ie, grade I or II), this may be a relative indication for surgical intervention to correct any causative factors, such as meniscal deficiency or overlying femoral condyle cartilage defects because the treatment of these defects is more difficult when they become full thickness. Proposed interventions include microfracture plus a scaffold, minced allograft cartilage, and retrograde drilling and implantation of an osteochondral allograft. In addition, the presence of underlying symptomatic bony lesions with corresponding subchondral bone marrow edema may warrant consideration of treatment with an injection of calcium phosphate to strengthen the subchondral bone.[27] This can be performed with image-guided injection of osteoconductive calcium phosphate bone substitute in the location of bone marrow edema, the therapeutic target. Using this technique for the treatment of symptomatic subchondral defects requires much additional research but has the potential to be used to treat patients with multiple types of subchondral bone injury, such as osteochondritis dissecans, osteoarthritis, and spontaneous osteonecrosis.

Other Factors

Several studies have attempted to correlate various factors with results of cartilage repair and regenerative procedures. Activity level has been used in a well-published algorithm for determining the best treatment for cartilage defects of varying size.[40,41] Age is another factor; patients younger than age 30 years have the best results;[42-44] however, this does not mean that patients older than age 30 years should not have cartilage restoration procedures. When the alternative is a joint arthroplasty, many patients, in an attempt to preserve their native joint and potentially avoid the risks associated with revision arthroplasty later in life, choose cartilage restoration despite slightly inferior results compared with younger patients with similar injuries. Also, a higher BMI has been associated with poorer results after microfracture[45] but has not been evaluated as a risk factor for inferior results with other cartilage restoration or transplantation techniques. Other significant factors to consider include the duration of symptoms, number of prior surgeries, smoking status, and gender.[46]

Surgical Indications

Indications for cartilage restoration include symptomatic, focal, International Cartilage Repair Society grade III or IV cartilage defects. Because

cartilage defects can be asymptomatic, the physician must determine whether the cartilage defect is the source of the patient's symptoms. Deficient meniscus or ligamentous restraints and any malalignment or overloading of the affected compartment should be addressed before or at the same time as the cartilage restoration procedure. Typically, patients have failed to respond to nonsurgical management and may have undergone a diagnostic arthroscopy and chondroplasty of the defect with detailed débridement of the lesion, creating vertical walls and possibly rendering the defect asymptomatic. Autologous chondrocyte implantation biopsy can be performed at this time as well, if indicated.

Summary

Cartilage defects are more prevalent than originally thought, and yet the natural history or what makes certain defects symptomatic is still not fully understood. Before treatment of a lesion, a careful history and physical examination is required to ensure that the patient's symptoms are actually related to the defect. Other factors must be considered to improve patient outcomes, including the status of the meniscus, assessing and treating malalignment or offloading the patellofemoral compartment, and reconstructing any ligamentous deficiencies. Understanding the clinical indications for cartilage restoration surgery and addressing all factors that may impact outcome are essential to the treatment of articular cartilage injury, regardless of the technique used to restore the injured cartilage or osteochondral unit.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 7, 8, 35, 38, 39, and 44 are level I studies. Reference 42 is a level II study. References 5 and 9 are level III studies. References 10, 11, 15-18, 23, 24, 28, 30, 43, and 45 are level IV studies. References 2, 4, 6, 12, 13, 25, 27, 29, 31, 36, 37, 40, 41, and 46 are level V expert opinion.

References printed in **bold type** are those published within the past 5 years.

1. Bear DM, Szczodry M, Kramer S, Coyle CH, Smolinski P, Chu CR: Optical coherence tomography detection of subclinical traumatic cartilage injury. *J Orthop Trauma* 2010;24(9):577-582.
2. Gomoll AH, Madry H, Knutsen G, et al: The subchondral bone in articular cartilage repair: Current problems in the surgical management. *Knee Surg Sports Traumatol Arthrosc* 2010;18(4):434-447.
3. Alford JW, Cole BJ: Cartilage restoration, part 1: Basic science, historical perspective, patient evaluation, and treatment options. *Am J Sports Med* 2005;33(2):295-306.
4. Messner K, Maletius W: The long-term prognosis for severe damage to weight-bearing cartilage in the knee: A 14-year clinical and radiographic follow-up in 28 young athletes. *Acta Orthop Scand* 1996;67(2):165-168.
5. Shelbourne KD, Jari S, Gray T: Outcome of untreated traumatic articular cartilage defects of the knee: A natural history study. *J Bone Joint Surg Am* 2003;85(suppl 2):8-16.
6. **Henn RF III, Gomoll AH: A review of the evaluation and management of cartilage defects in the knee. *Phys Sportsmed* 2011;39(1):101-107.**
7. **Crawford DC, DeBerardino TM, Williams RJ III: NeoCart, an autologous cartilage tissue implant, compared with microfracture for treatment of distal femoral cartilage lesions: An FDA phase-II prospective, randomized clinical trial after two years. *J Bone Joint Surg Am* 2012;94(11):979-989.**
8. **Stanish WD, McCormack R, Forriol F, et al: Novel scaffold-based BST-CarGel treatment results in superior cartilage repair compared with microfracture in a randomized controlled trial. *J Bone Joint Surg Am* 2013;95(18):1640-1650.**
9. Arøen A, Løken S, Heir S, et al: Articular cartilage lesions in 993 consecutive knee arthroscopies. *Am J Sports Med* 2004;32(1):211-215.
10. Flanigan DC, Harris JD, Trinh TQ, Siston RA, Brophy RH: Prevalence of chondral defects in athletes' knees: A systematic review. *Med Sci Sports Exerc* 2010;42(10):1795-1801.
11. Peterson L, Minas T, Brittberg M, Lindahl A: Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation: Results at two to ten years. *J Bone Joint Surg Am* 2003;85(suppl 2):17-24.
12. Gomoll AH, Farr J, Gillogly SD, Kercher J, Minas T: Surgical management of articular cartilage defects of the knee. *J Bone Joint Surg Am* 2010;92(14):2470-2490.
13. Collado H, Fredericson M: Patellofemoral pain syndrome. *Clin Sports Med* 2010;29(3):379-398.
14. **Efe T, Füglein A, Getgood A, et al: Anterior cruciate ligament deficiency leads to early instability of scaffold for cartilage regeneration: A controlled laboratory ex-vivo study. *Int Orthop* 2012;36(6):1315-1320.**
15. Kocher MS, DiCanzio J, Zurakowski D, Micheli LJ: Diagnostic performance of clinical examination and selective magnetic resonance imaging in the evaluation of intraarticular knee disorders in children and adolescents. *Am J Sports Med* 2001;29(3):292-296.
16. Fleming BC, Oksendahl HL, Mehan WA, et al: Delayed Gadolinium-Enhanced MR Imaging of Cartilage (dGEMRIC) following ACL injury. *Osteoarthritis Cartilage* 2010;18(5):662-667.
17. Strobel MJ, Weiler A, Schulz MS, Russe K, Eichhorn HJ: Arthroscopic evaluation of articular cartilage lesions in posterior-cruciate-ligament-deficient knees. *Arthroscopy* 2003;19(3):262-268.
18. Corten K, Bellemans J: Cartilage damage determines intermediate outcome in the late multiple ligament and posterolateral corner-reconstructed knee: A 5- to 10-year follow-up study. *Am J Sports Med* 2008;36(2):267-275.
19. Fukubayashi T, Kurosawa H: The contact area and pressure distribution pattern of the knee: A study of normal and osteoarthrotic knee joints. *Acta Orthop Scand* 1980;51(6):871-879.
20. Seedhom BB, Hargreaves DJ: Transmission of load in the knee joint with special reference to the role of the menisci. *Eng Med* 1979;8(4):220-228.
21. Verma NN, Kolb E, Cole BJ, et al: The effects of medial meniscal transplantation techniques on intra-articular contact pressures. *J Knee Surg* 2008;21(1):20-26.
22. Macconaill MA: The function of the intra-articular fibrocartilages, with special reference to the knee and inferior radioulnar joints. *J Anat* 1932;66(Pt 2):210-227.
23. 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(9):1911-1920.

24. Behery OA, Harris JD, Karnes JM, Siston RA, Flanigan DC: Factors influencing the outcome of autologous chondrocyte implantation: A systematic review. *J Knee Surg* 2013;26(3):203-211.

25. Brandt KD, Radin EL, Dieppe PA, van de Putte L: Yet more evidence that osteoarthritis is not a cartilage disease. *Ann Rheum Dis* 2006;65(10):1261-1264.

26. Armstrong CG: An analysis of the stresses in a thin layer of articular cartilage in a synovial joint. *Eng Med* 1986;15(2):55-61.

27. Abrams GD, Alentorn-Geli E, Harris JD, Cole BJ: Treatment of a lateral tibial plateau osteochondritis dissecans lesion with subchondral injection of calcium phosphate. *Arthrosc Tech* 2013;2(3):e271-e274.

28. Bartlett W, Gooding CR, Carrington RW, Skinner JA, Briggs TW, Bentley G: Autologous chondrocyte implantation at the knee using a bilayer collagen membrane with bone graft: A preliminary report. *J Bone Joint Surg Br* 2005;87(3):330-332.

29. Dugdale TW, Noyes FR, Styer D: Preoperative planning for high tibial osteotomy: The effect of lateral tibiofemoral separation and tibiofemoral length. *Clin Orthop Relat Res* 1992;274:248-264.

30. Trinh TQ, Harris JD, Siston RA, Flanigan DC: Improved outcomes with combined autologous chondrocyte implantation and patellofemoral osteotomy versus isolated autologous chondrocyte implantation. *Arthroscopy* 2013;29(3):566-574.

31. Farr J, Schepsis A, Cole B, Fulkerson J, Lewis P: Anteromedialization: Review and technique. *J Knee Surg* 2007;20(2):120-128.

32. Rue JP, Colton A, Zare SM, et al: Trochlear contact pressures after straight anteriorization of the tibial tuberosity. *Am J Sports Med* 2008;36(10):1953-1959.

33. Beck PR, Thomas AL, Farr J, Lewis PB, Cole BJ: Trochlear contact pressures after anteromedialization of the tibial tubercle. *Am J Sports Med* 2005;33(11):1710-1715.

34. Guettler JH, Demetropoulos CK, Yang KH, Jurist KA: Osteochondral defects in the human knee: Influence of defect size on cartilage rim stress and load redistribution to surrounding cartilage. *Am J Sports Med* 2004;32(6):1451-1458.

35. 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(9):1066-1075.

36. Abrams GD, Mall NA, Fortier LA, Roller BL, Cole BJ: BioCartilage: Background and operative technique. *Oper Tech Sports Med* 2013;21(2):116-124.

37. Kon E, Filardo G, Roffi A, Andriolo L, Marcacci M: New trends for knee cartilage regeneration: From cell-free scaffolds to mesenchymal stem cells. *Curr Rev Musculoskelet Med* 2012;5(3):236-243.

38. Saris DB, Vanlauwe J, Victor J, et al: Characterized chondrocyte implantation results in better structural repair when treating symptomatic cartilage defects of the knee in a randomized controlled trial versus microfracture. *Am J Sports Med* 2008;36(2):235-246.

39. Vanlauwe J, Saris DB, Victor J, Almqvist KF, Bellemans J, Luyten FP; TIG/ACT/01/2000&EXT Study Group: Five-year outcome of characterized chondrocyte implantation versus microfracture for symptomatic cartilage defects of the knee: Early treatment matters. *Am J Sports Med* 2011;39(12):2566-2574.

40. Cole BJ, Pascual-Garrido C, Grumet RC: Surgical management of articular cartilage defects in the knee. *J Bone Joint Surg Am* 2009;91(7):1778-1790.

41. Cole BJ, Pascual-Garrido C, Grumet RC: Surgical management of articular cartilage defects in the knee. *Instr Course Lect* 2010;59:181-204.

42. Bekkers JE, Inklaar M, Saris DB: Treatment selection in articular cartilage lesions of the knee: A systematic review. *Am J Sports Med* 2009;37(suppl 1):148S-155S.

43. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG: Outcomes of microfracture for traumatic chondral defects of the knee: Average 11-year follow-up. *Arthroscopy* 2003;19(5):477-484.

44. 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(3):455-464.

45. Mithoefer K, McAdams T, Williams RJ, Kreuz PC, Mandelbaum BR: Clinical efficacy of the microfracture technique for articular cartilage repair in the knee: An evidence-based systematic analysis. *Am J Sports Med* 2009;37(10):2053-2063.

46. Behery O, Siston RA, Harris JD, Flanigan DC: Treatment of cartilage defects of the knee: Expanding on the existing algorithm. *Clin J Sport Med* 2014;24(1):21-30.