

Combined Cartilage Restoration and Distal Realignment for Patellar and Trochlear Chondral Lesions

Peter N. Chalmers, MD; Adam B. Yanke, MD; Vasili Karas, BS; Seth L. Sherman, MD; Brian J. Cole, MD

American Academy of Orthopaedic Surgeons Web site: *Orthopaedic Knowledge Online* 2012 10(1): http://orthopaortal.aaos.org/oko/article.aspx?article=OKO_SPO069. Accessed January 1, 2012

©2001 - 2011 by the American Academy of Orthopaedic Surgeons. "All Rights Reserved."
This website and its contents may not be reproduced in whole or in part without written permission.

Pathophysiology and Etiology

Articular cartilage defects of the patellofemoral (PF) joint are relatively common, although the exact incidence and natural history of such lesions are poorly understood. Widuchowski et al¹ reported a 60% incidence of PF lesions in a series of more than 25,000 knee arthroscopies performed in patients with knee pain due to a variety of etiologies. Of the lesions reported, 82% were patellar lesions and 18% were trochlear. In another series, this of National Basketball Association players without knee pain, 12 of 28 knees (44%) were found to have patellar chondral changes on MRI, while 7 of 28 (26%) were found to have trochlear chondral changes.²

As cartilage is aneural, patients' discomfort may originate from the subchondral bone or peripatellar synovial tissue that is compressed during lateral patellar subluxation. However, why certain PF lesions become symptomatic while others remain subclinical is poorly understood. The most notable factors contributing to the incidence of osteochondral defects of the PF joint include PF instability and increased contact stress. Although no study has directly correlated the presence of PF cartilage lesions with instability, the incidence of such lesions in patients with a history of dislocation has been reported to be up to 95%.³ Bony aspects of knee anatomy that predispose the patella to lateral instability include anatomic valgus of the knee, increased quadriceps angle (Q angle) in females as a result of gynecoid pelvic morphology, altered tibial tubercle-to-trochlear groove (TT-TG) distance, and relative hypoplasia of the lateral patellar facet or trochlear dysplasia. Soft-tissue pathology that can accentuate this predisposition includes lateral retinacular tightness, medial retinacular laxity, and laxity of the medial patellofemoral ligament (MPFL). In addition to acute traumatic injuries such as direct loading or dislocation of the patella, chronic PF joint injury can accumulate through microtrauma from repetitive loading.

Articular cartilage degeneration in the PF joint may be the result of high contact stresses, which range from more than 3 times body weight during stair climbing to 20 times body weight with jumping. PF joint stress is determined by the forces applied to the patella from the pull of the quadriceps and patellar tendons, the MPFL, and the lateral retinaculum divided by the area of contact between the trochlea and patella. Force mismatch may decrease joint contact area by loss of PF joint congruency,⁴ which can occur as a result of excess lateral pull, rupture of the MPFL, contracture of the lateral retinaculum, or by lateral pull of the quadriceps or patellar tendons. Articular cartilage lesions may increase PF joint stress by decreasing available joint contact area. Thus, the symptomatic efficacy of any procedure used in the treatment of PF chondral defects may lie in providing relief of these contact stresses and, possibly, by removing excess strain on the exquisitely innervated synovial tissues as well.

Anteromedialization of the tibial tubercle (AMZ) improves PF joint contact area and decreases the quadriceps advantage by recentering the patella in the trochlea, resulting in the unloading of irregular lateral patellar and trochlear defects. Cartilage restoration may aid this process by decreasing the inflammatory stimulus and resulting synovial irritation from cartilage defects. These biomechanical changes have been demonstrated by several cadaveric studies. In a study of 10 cadaveric knees, AMZ significantly decreased the mean total contact pressure while shifting contact pressure toward the medial trochlea.⁵ In a similar study, straight anteriorization of the tibial tubercle was shown to decrease peak and mean joint pressures at 30° and 60° of knee flexion.⁶ Anteriorization may be doubly useful in that the resulting decrease in joint reaction force could synergistically affect joint contact area.⁷

In addition to altering PF joint mechanics, AMZ alters tibiofemoral mechanics by increasing varus angulation and tibial external rotation and posterior shift.⁸ The long-term effects of such changes on the tibiofemoral cartilage are unknown, but these alterations may undermine the goal of the AMZ procedure.

Diagnosis

Patient History

A comprehensive history and physical examination are critical for obtaining the proper diagnosis and determining the treatment plan for articular cartilage defects of the PF joint. Anterior knee pain, either at or surrounding the patella, is the most common complaint; however, posterior knee pain may also suggest a trochlear defect. Activities that exacerbate anterior knee pain, past traumatic events to the knee, and a full accounting of past therapies attempted should also be evaluated. Activities that typically exacerbate pain related to PF cartilage lesions include running, squatting, jumping, kneeling, and stair climbing, although any activity with prolonged or repetitive knee flexion can cause pain. Commonly, symptoms are exacerbated during sporting activities and are associated with increased swelling. Subjective symptoms of crepitus, locking, giving-way, or catching with knee flexion may signal underlying cartilage lesions, but such symptoms are nonspecific. Although no organized trials have been conducted to determine the sensitivity and specificity of various historical findings associated with PF cartilage defects, one of the strongest predictors is a history of dislocation. In one series, articular cartilage defects were found in 95% of knees with initial lateral patellar dislocation.³

Physical Examination

Although no specific combination of findings has been associated with a high likelihood of an underlying PF cartilage defect, the physical examination of patients with anterior knee pain should include gait analysis, assessment of alignment, tests for muscle imbalance, and provocative maneuvers. The practitioner should look for dynamic and static conditions that increase lateral stress on the PF joint, including an in-toe gait, hip abductor weakness, contracture of the iliotibial band, increased femoral anteversion, and valgus malalignment. When the patient is asked to stand on the affected leg and raise the contralateral limb, a pelvic dip may be noted on the contralateral side (Trendelenburg sign), which is indicative of hip abductor weakness.

Though its value has been recently questioned, the Q angle is determined during physical examination by measuring the angle from the anterior superior iliac spine to the center of the patella to the tibial tubercle (Figure 1). A normal value for the Q angle in a male is 14° ±3°, whereas in a female the normal value is 17° ±3°. As the patella may already be subluxated in extension, because it is not engaged in the trochlea, examination should also be carried out at 30° of knee flexion. The Q angle, however, is highly variable because it depends on several dynamic and static factors, and interobserver reliability of this measurement is poor as well. Because of these factors, the Q angle has largely been replaced by objective measures of the TT-TG distance.



Fig. 1

A weight-bearing, full-length radiograph of the lower extremity can be used to calculate the Q angle, which is the angle formed between the lines of pull of the quadriceps (green line from the anterior superior iliac spine to the center of the patella) and the patellar (red line from the center of the patella to the tibial tubercle) tendons.

Muscular imbalance, including vastus medialis wasting, may also be present with prolonged symptoms. Specific examinations should include testing for tenderness to palpation in the peripatellar soft tissues, patellar mobility at 0°, 30°, and 60° of knee flexion, patellar tilt, apprehension with a lateral patellar force applied at 30° of knee flexion, and testing for the so-called J-sign as the patella dislocates laterally out of the trochlea with extension. The latter test is slightly more specific for MPFL laxity versus lateral contracture.¹⁰

To test for pain secondary to PF loading, the half-squat test can be performed, which is positive when holding a squatting position recreates the anterior knee pain. The sensitivity and specificity of this test are unknown, however.⁹

Imaging

Standard radiographic views of the knee, including AP, lateral, Rosenberg (PA view in 45° of flexion), and Merchant views, can be helpful in ruling out fracture, osteoarthritis, inflammatory arthritis, and loose osteochondral bodies. Such views are also helpful in assessing for trochlear dysplasia, patellar tilt, and patellar subluxation (Figure 2).

Fig. 2

Radiographic views used in the evaluation of patients with patellofemoral cartilage defects.



Fig. 2A

AP view.



Fig. 2B

Rosenberg view (PA view in 45° of flexion).

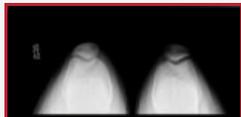


Fig. 2C

Merchant (axial) view.



Fig. 2D

Lateral view.

The weight-bearing mechanical axis view on a long cassette can be used to calculate the Q angle and to assess tibiofemoral alignment (see Figure 1). However, significant interobserver variability and anatomic variation have been described for this measurement, which can significantly complicate interpretation.¹⁰ A perfect lateral view can also be useful in comparing the eminence of the medial and lateral condyles, aiding in identification of trochlear dysplasia. The Merchant view is useful in evaluating for PF osteoarthritis

and joint-space narrowing, which is commonly seen at 45° of flexion, but is less useful for assessing for patellar maltracking and condylar dysplasia, which are seen more prominently at 30° of flexion.¹⁰

CT and/or MRI are often more useful for evaluation of patients with PF cartilage defects. Although these advanced imaging studies are not used in all patients, they are especially important in obese patients for whom accurate clinical examination can be difficult. A CT scan can more precisely define patellar and trochlear osseous anatomy than can a Merchant view because the entire trochlea can be assessed. The TT-TG distance is determined by superimposing an axial cut through the PF joint over another one through the tibial tubercle, and then measuring the length of a line drawn in the coronal plane between the tubercle and the deepest portion of the trochlear groove (Figure 3).¹⁰ A TT-TG distance of 15 mm is considered normal, and values of 20 mm or greater indicate likely patellar maltracking. More than 50% of patients with patellar maltracking have a TT-TG distance of greater than 20 mm, whereas this is true in less than 5% of asymptomatic knees.¹¹

Fig. 3

Calculation of tibial tubercle-to-trochlear groove (TT-TG) distance. This distance is determined by superimposing an axial CT scan through the patellofemoral joint over one through the tibial tubercle, and then measuring the length of a line drawn in the coronal plane between the tubercle and the deepest portion of the trochlear groove. A TT-TG distance of 15 mm is considered normal, and a value of 20 mm or greater is an indication for anteromedialization of the tibial tubercle.

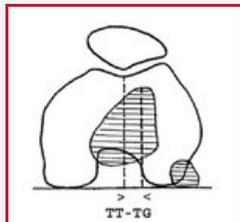


Fig. 3A

Schematic representation of TT-TG distance calculation.

(Reproduced with permission from Gomoll AH, Minas T, Farr J, Cole BJ: Treatment of chondral defects in the patellofemoral joint. *J Knee Surg* 2006;19(4): 285-295.)



Fig. 3B

Superimposition of axial CT scans for determination of TT-TG distance.

(Reproduced with permission from Gomoll AH, Minas T, Farr J, Cole BJ: Treatment of chondral defects in the patellofemoral joint. *J Knee Surg* 2006;19(4): 285-295.)

TT-TG distance has been shown to be superior to Q angle for assessing patellar tracking and, with a proper CT-based protocol, use of this measurement exposes the patient to minimal additional radiation.¹² MRI can also be used to calculate the TT-TG distance, with an accuracy equivalent to that of CT.¹³ The sensitivity and specificity of MRI for cartilage lesions can exceed 90%. However, the diagnosis of an osteochondral or chondral defect on MRI does not establish causality between these lesions and a patient's symptoms. Therefore, the patient history and physical examination remain of primary importance in the diagnosis of symptomatic PF articular cartilage defects.

Differential Diagnosis

The differential diagnosis for anterior knee pain is broad and includes PF maltracking, patellar articular cartilage defects, trochlear articular cartilage defects, meniscal tears, ligamentous injury, patellar tendinitis, plicae, prepatellar bursitis, pathology associated with the hip joint or ankle joint, infectious causes, neoplastic causes, and vascular causes. In addition, the psychiatric and social histories may be illuminating in patients for whom no physical abnormality can be detected.

Management Options

Therapies for articular cartilage defects of the PF joint are aimed at unloading the diseased cartilage and relieving stress upon the peripatellar soft tissues. A variety of nonsurgical treatments, some performed in conjunction with a physical therapist, have been successfully used in the treatment of PF articular cartilage defects, and should be attempted prior to surgical intervention. Physical therapy should include quadriceps stretching; mobilization of the peripatellar soft tissues; strengthening of the core musculature, vastus medialis obliquus (VMO) and hip abductors; closed-chain exercises; taping; bracing; cooling therapy; use of NSAIDs; and gait training. These treatments should be used for at least 6 months while monitoring for return of VMO function and elimination of the Trendelenburg gait. If these treatments fail to relieve symptoms, surgical intervention can be considered. Office assessment that includes establishing the patient's ability to perform single-leg tasks can help to define residual weakness of core musculature. This must be corrected prior to consideration for surgical intervention unless the pathology is so profound that it completely inhibits the patient's ability to regain this dynamic function.

If nonsurgical treatment fails to improve a patient's symptoms, a number of surgical options exist, including proximal realignment of the quadriceps tendon, VMO advancement, MPFL reconstruction, medial retinacular plication, lateral retinacular release, and distal realignment with osteotomy of the tibial tubercle. To address the cartilaginous lesion(s), procedures such as autologous chondrocyte implantation (ACI) or PF arthroplasty may be implemented. Each procedure has a role for a specific patient population, and choosing which procedure to perform requires experience and judgment on the part of the surgeon. Arthroscopy is of particular importance in selecting a surgical plan, and is the standard for characterizing lesion pathology on the trochlea or patella. Arthroscopy also allows biopsy for ACI, should the surgeon choose to select this procedure.

Cartilage Restoration

Options for restoring articular cartilage are limited by the ability to replicate the complex three-dimensional anatomy of the PF joint.^{10,12,14,15,16} The decision of which cartilage restoration procedure to pursue depends on the size of the lesion, the level of demand the patient places on his/her knees, and whether a previous attempt at cartilage restoration

has failed.

Microfracture

In general, microfracture is the first-line therapy for low-demand patients and for smaller lesions. Though short-term results of microfracture of trochlear and patellar lesions have been acceptable, these results have deteriorated at midterm and long-term follow-up.^{17,18} The mechanical properties of the fibrocartilage created by microfracture may be poorly suited to the PF joint because of the high shear stresses. Caution in interpreting the literature describing the outcomes of microfracture is warranted. Because microfracture is relatively easy to perform and is often the first-line treatment for a symptomatic cartilage defect, comorbidities such as malalignment are often neglected at this early decision-making stage. If all the principles of comorbidity correction are adhered to, however, it is entirely possible that microfracture could perform more optimally in this patient population.

Osteochondral Allograft Transplantation

Osteochondral allograft transplantation has also been used in the PF joint, although previous attempts have been limited to diffuse lesions as a salvage procedure. The complex topology of the PF joint and the requirement for high congruence have limited past attempts.^{19,20,21} Overall, results of osteochondral allograft transplantation have not been as successful for lesions on PF joint surfaces as they have been for lesions on the femoral condyles, but in some clinical scenarios osteochondral allograft transplantation remains an early treatment option.^{19,20,21}

Autologous Chondrocyte Implantation

ACI is preferred for high-demand patients, patients with large lesions, and for those who have undergone a failed previous cartilage restoration attempt. ACI has been extensively studied, with the largest and most well-conducted trials of any cartilage restoration procedure used in the PF joint, and has the additional benefit of "self-conforming" to the topography of the joint.^{15,16,22,23}

Contraindications to ACI in the PF joint include inflammatory arthritis, loss of subchondral bone, disease within the tibiofemoral joint, and a high likelihood of patient noncompliance with postoperative rehabilitation and restrictions. The potential benefits of ACI must be juxtaposed against the disadvantages, which include a high revision rate for débridement (33% for trochlear lesions).²³ A bipolar lesion such as a "kissing lesion" is a relative contraindication to cartilage restoration and would likely benefit from PF arthroplasty. Notably, the complication rate for ACI, including the need for reoperation, has dramatically decreased as a result of the off-label usage of a synthetic collagen I/III membrane as an alternative to periosteum, as described by Gomoll et al.²⁴

Anteromedialization of the Tibial Tubercle

AMZ is the most commonly used realignment procedure in the treatment of PF cartilage defects. The patients who are most likely to benefit from AMZ are those with (1) symptomatic lesions, (2) patellar maltracking, and (3) lesions that can be unloaded onto healthy cartilage through tibial tubercle osteotomy. Numerous authors have recommended AMZ, either alone or in association with a cartilage restoration procedure, if these three conditions are met.^{12,16,25}

Prior to osteotomy, surgeons should document patellar maltracking via abnormal TT-TG values whenever possible, as well as healthy medial trochlear cartilage and, if possible, central trochlear and patellar cartilage as well. Symptoms must also be attributed to maltracking, otherwise the interventions upon the resulting articular cartilage lesions will fail to improve the patient's outcome. In addition, while AMZ may work well for patients with articular cartilage defects and early-stage degeneration, advanced osteoarthritis may signal that this procedure is less likely to succeed, primarily because of the lack of healthy cartilage onto which the diseased cartilage can be unloaded. These patients may be better served by PF arthroplasty or total knee arthroplasty, depending on the condition of the tibiofemoral cartilage. The AMZ procedure itself can be customized to unload the diseased cartilage by changing the slope of the osteotomy to provide more anteriorization in patients with central or proximal lesions, or more medialization in patients with lateral lesions.^{5,6,16} If chondral lesions are proximal and medial, AMZ alone is contraindicated, and may worsen symptoms because the forces would be transferred to this area.

Benefits of AMZ must be weighed against the additional risks of infection, symptomatic hardware, wound complications, nonunion (especially in obese patients, diabetic patients, and smokers), tibial fracture, compartment syndrome, and deep vein thrombosis. Removal of hardware may be required in up to 50% of patients.²² Several series have reported tibial fractures with AMZ, recommending extended weight-bearing limitations.²⁶

Treatment Algorithm

Considering all of these factors regarding cartilage restoration and realignment, we have developed an algorithm for the treatment of PF chondral lesions (Figure 4). In low-demand patients, microfracture should be considered, whereas in higher-demand patients, ACI should be considered. There is less definitive evidence to support osteochondral allograft transplantation or autograft transplantation. For larger lesions, ACI and allografting should be more strongly considered. AMZ should be considered in conjunction with ACI, osteochondral allograft transplantation, or autograft transplantation, especially for patients with symptomatic maltracking and healthy cartilage onto which the repair can be offloaded. Patients who have failed a previous cartilage restoration with microfracture may be most appropriately treated with ACI in conjunction with AMZ, and those who have failed ACI may be best treated with osteochondral allograft transplantation in conjunction with AMZ.



Fig. 4

Algorithm for treatment of patellofemoral osteochondral defects.

Exposure

The patient is positioned supine with an optional tourniquet in place, which can facilitate exposure and visibility. The size of the skin incision will vary depending on whether a concomitant cartilage procedure is performed. After incising skin, the soft tissue about the tibial tubercle is released to allow mobilization. The lateral soft-tissue dissection is extended to the tibial tubercle and crest, allowing for blunt elevation of the anterior compartment contents. After lateral exposure to the posterior surface of the tibia, a retractor is placed posteriorly to protect the anterior tibial artery and the deep peroneal nerve (Figure 5).

Fig. 5

Intraoperative photographs of a jig used for the anteromedialization of the tibial tubercle procedure.



Fig. 5A

Proximal pin guide.

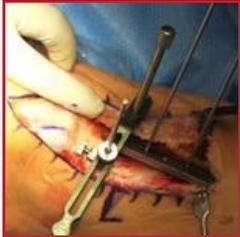


Fig. 5B

60° guide connected to jig.



Fig. 5C

Cutting through jig with a retractor placed posteriorly to protect the anterior tibial artery and the deep peroneal nerve.



Video

Combined Cartilage Restoration and Distal Realignment for Patellofemoral Cartilage Lesions: Complete video

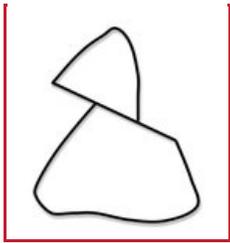
(touch graphic to view video)

Tibial Tubercle Osteotomy

Once appropriate exposure has been achieved, the anterior proximal aspect of the tibial tubercle osteotomy is done medial to the tubercle and progresses laterally, exiting the lateral cortex approximately 7 to 10 cm distal and relatively anterior to decrease stress risers. Our preference is to carry out the osteotomy with a jig that allows specific control of the coronal aspect of the proximal cut, rotation in the axial plane, and visualization of the distal exit point. Using this system, a 60° slope will yield 1 mm of medialization per 2 mm of elevation. Modifying the slope is important because the amount of medialization preferred is more widely varied (Figure 6). Therefore, given 1.0 to 1.5 cm of anteriorization, the slope should be adjusted to yield the appropriate amount of medialization with this setting. After anchoring the jig, an oscillating saw is used to complete the cut, and the jig is then removed. An osteotome and freehand saw are used to finish the cuts proximally, allowing rotation about the distally attached segment. This can be provisionally fixed using a Kirschner wire, with final fixation obtained using two 4.5-mm cortical screws.

Fig. 6

Schematic of an axial view of the proximal tibial metaphysis following tibial tubercle osteotomy, with translation of the tubercle fragment anteriorly and medially.



Cartilage Restoration

Attention is then directed to the PF joint for cartilage restoration. The microfracture and osteochondral allograft transplantation procedures have been reviewed elsewhere within *Orthopaedic Knowledge Online*, and we refer the reader to these papers. As our technique for ACI differs slightly from that published previously, it is briefly reviewed here.

For this procedure, cartilage biopsies must be harvested 6 to 12 weeks prior to implantation. During this prior procedure, chondral biopsies (200 to 300 μg) are obtained arthroscopically from the intercondylar notch and sent to a laboratory for expansion. During the implantation procedure, the patella is exposed through a lateral parapatellar incision. The patella is everted 90° to expose the cartilaginous undersurface. The cartilage lesion must be meticulously prepared with a scalpel and curet by removing all diseased cartilage without violation of the subchondral plate (Figures 7 and 8). The edges of the lesion must also be sculpted to form vertical walls of healthy articular cartilage. The tourniquet should be let down prior to proceeding with the next step. Meticulous hemostasis must be obtained. In general, we do not recommend flipping the tibial tubercle proximally because this requires more extensive dissection around the fat pad, which leads to more postoperative scar formation and, potentially, to problems associated with stiffness and arthrofibrosis.



Fig. 7

Intraoperative photograph showing a diffuse Pidoriano type IV patellar cartilage lesion.



Fig. 8

Intraoperative photograph of the lesion in Figure 7, with all diseased cartilage removed, the subchondral plate left intact, and with vertical walls of healthy articular cartilage.

The defect can then be covered with the off-label use of a synthetic collagen matrix (Bio-Gide; Geistlich Pharma AG, Wolhusen, Switzerland), which we prefer over a periosteal patch so as to reduce postoperative graft hypertrophy. We cannot, however, recommend this product for this indication, as it has not been approved by the US Food and Drug Administration for this use.²⁴ This patch is then sutured with dyed 6-0 Vicryl (Ethicon, Somerville, NJ). The sutures should be spaced 2 to 3 mm apart, leaving a gap superiorly to allow insertion of the angiocath for chondrocyte implantation. The suture line is then reinforced with fibrin glue, and its integrity to water pressure is tested to assure a watertight seal. After injection of chondrocyte suspension from a tuberculin syringe, the remaining gap is affixed with sutures and fibrin glue (Figure 9). Recently, we have begun soaking the patch in the contents of one cell vial and using the second vial after the patch is secured as the injectable suspension.



Fig. 9

Intraoperative image of the lesion in Figures 7 and 8, with the sutured and fibrin-glued patch in place, receiving cultured chondrocytes.

Closure

The arthrotomy should be closed from proximal to distal, ensuring appropriate balance of the patella in the trochlea. The lateral release site may be left open or simply lengthened, as dictated by patellar tracking and soft-tissue tension at the time of surgery.

Outcomes

Outcomes of Isolated Procedures

Outcomes have historically been poor for AMZ and cartilage restoration procedures performed in isolation. In a retrospective study, Pidioriano et al²⁶ correlated patellar articular cartilage lesions with the outcomes of AMZ. Whereas 87% of patients with lateral or distal patellar lesions had excellent or good outcomes, only 55% of those with central lesions had good or excellent outcomes, and only 20% of those with proximal or diffuse lesions had good or excellent outcomes (Figure 10).

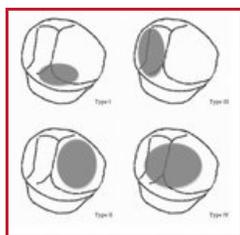


Fig. 10

Four patellar zones as described by Pidioriano et al: distal (type I), lateral (II), medial (III), and diffuse (IV). Patients with lateral and distal patellar lesions had overall excellent or good outcomes with anteromedialization of the tibial tubercle, whereas only 20% of those with proximal or diffuse lesions had good or excellent outcomes.

Similar results have been shown for ACI procedures used in isolation. Brittberg et al²⁷ described intact articular cartilage at the site of ACI in only one of seven patellar lesions (14%). The same researchers concluded that concomitant realignment would likely improve the results. Jamali et al²⁰ and Torga Spak and Teitge²¹ in small series reported 25% and 42% conversion to arthroplasty, respectively, after osteochondral allografting. Kreuz et al¹⁷ noted significantly worse outcomes with microfracture performed in the PF compartment compared with the tibiofemoral joint, recommending against this form of cartilage restoration. Bentley et al¹⁹ in a randomized comparative trial between ACI and mosaicplasty, noted a trend toward inferior outcomes in the underpowered PF subgroup, which they attributed to differences in thickness and matrix orientation between donor and recipient cartilage.

Outcomes of Combination Procedures

Adding AMZ to a concomitant cartilage restoration procedure has resulted in superior outcomes in several series. In a retrospective comparative trial of ACI, Pascual-Garrido et al¹⁶ demonstrated a trend toward better outcomes in patients who underwent AMZ, most notably in patient satisfaction scores. In this study, 86% of patients receiving ACI and AMZ were mostly to completely satisfied, whereas only 45% of those who received ACI alone reported the same results.

Excellent results have been demonstrated in patients receiving ACI and AMZ for lateral PF lesions.²³ Mandelbaum et al²⁸ reported excellent outcomes in patients who underwent ACI for trochlear lesions, 41% of whom had concomitant or previous AMZ, with significant differences between combination procedures and ACI alone. Minas and Bryant¹⁵ demonstrated excellent outcomes among 31 central or diffuse patellar lesions treated with AMZ and ACI, a group directly comparable with those Pidioriano et al²⁶ characterized as having the worst outcomes. They attributed the relative success in this group of patients to the combination of AMZ and ACI.

AAOS Clinical Practice Guidelines

The most recent AAOS clinical practice guideline for nonarthroplasty treatment of osteoarthritis of the knee is unable to make recommendations for or against AMZ.²⁹ (Click [here](#) for a summary of the guideline.) At the time of this publication, no AAOS guideline on the treatment of articular cartilage defects in the knee has been released.

Conclusion

Chondral lesions of the patellofemoral joint are relatively common among knee pathologies, and can cause significant anterior knee pain. A variety of procedures are available to treat these conditions. Deciding which combination of procedures to perform, as well as how to perform them, is complex and requires consideration of the patient's injury history, symptomatology, and pathoanatomy. In addition, patient expectations must be carefully managed because the length of time required to achieve satisfactory clinical improvement can approach 12 to 18 months in some instances. In this paper, we offer an algorithm to aid in surgical decision making. Though high-quality evidence is lacking, the use of combination procedures for PF articular cartilage defects has been shown to result in excellent outcomes, whereas AMZ or ACI performed in isolation have had traditionally poor results.

References

1. Widuchowski W, Widuchowski J, Trzaska T: Articular cartilage defects: Study of 25,124 knee arthroscopies. *Knee* 2007;14(3):177-182.
2. Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ: Abnormal findings on knee magnetic resonance imaging in asymptomatic NBA players. *J Knee Surg* 2008;21(1):27-33.
3. Nomura E, Inoue M, Kurimura M: Chondral and osteochondral injuries associated with acute patellar dislocation. *Arthroscopy* 2003;19(7):717-721.
4. Farr J, Schepsis A, Cole B, Fulkerson J, Lewis P: Anteromedialization: Review and technique. *J Knee Surg* 2007;20(2):120-128.
5. 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.
6. 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.
7. Goudakos IG, König C, Schöttle PB, et al: Regulation of the patellofemoral contact area: An essential mechanism in patellofemoral joint mechanics? *J Biomech* 2010;43(16):3237-3239.
8. Mani S, Kirkpatrick MS, Saranathan A, Smith LG, Cosgarea AJ, Elias JJ: Tibial tuberosity osteotomy for patellofemoral realignment alters tibiofemoral kinematics. *Am J Sports Med* 2011;39(5):1024-1031.
9. Mihalko WM, Boachie-Adjei Y, Spang JT, Fulkerson JP, Arendt EA, Saleh KJ: Controversies and techniques in the surgical management of patellofemoral arthritis. *Instr Course Lect* 2008;57:365-380.
10. Gomoll AH, Minas T, Farr J, Cole BJ: Treatment of chondral defects in the patellofemoral joint. *J Knee Surg* 2006;19(4):285-295.

11. Dejour H, Walch G, Nove-Josserand L, Guier C: Factors of patellar instability: An anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc* 1994;2(1):19-26.
12. Gallo RA, Feeley BT: Cartilage defects of the femoral trochlea. *Knee Surg Sports Traumatol Arthrosc* 2009;17(11):1316-1325.
13. Schoettle PB, Zanetti M, Seifert B, Pfirrmann CW, Fucentese SF, Romero J: The tibial tuberosity-trochlear groove distance: A comparative study between CT and MRI scanning. *Knee* 2006;13(1):26-31.
14. Henderson IJ, Lavigne P: Periosteal autologous chondrocyte implantation for patellar chondral defect in patients with normal and abnormal patellar tracking. *Knee* 2006;13(4):274-279.
15. Minas T, Bryant T: The role of autologous chondrocyte implantation in the patellofemoral joint. *Clin Orthop Relat Res* 2005;436:30-39.
16. Pascual-Garrido C, Slabaugh MA, L'Heureux DR, Friel NA, Cole BJ: Recommendations and treatment outcomes for patellofemoral articular cartilage defects with autologous chondrocyte implantation: Prospective evaluation at average 4-year follow-up. *Am J Sports Med* 2009;37(suppl 1):33S-41S.
17. Kreuz PC, Steinwachs MR, Ergelet C, et al: Results after microfracture of full-thickness chondral defects in different compartments in the knee. *Osteoarthritis Cartilage* 2006;14(11):1119-1125.
18. 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.
19. Bentley G, Biant LC, Carrington RW, et al: A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br* 2003;85(2):223-230.
20. Jamali AA, Emmerson BC, Chung C, Convery FR, Bugbee WD: Fresh osteochondral allografts: Results in the patellofemoral joint. *Clin Orthop Relat Res* 2005;437:176-185.
21. Torga Spak R, Teitge RA: Fresh osteochondral allografts for patellofemoral arthritis: Long-term followup. *Clin Orthop Relat Res* 2006;444:193-200.
22. Farr J: Autologous chondrocyte implantation improves patellofemoral cartilage treatment outcomes. *Clin Orthop Relat Res* 2007;463:187-194.
23. McNickle AG, L'Heureux DR, Yanke AB, Cole BJ: Outcomes of autologous chondrocyte implantation in a diverse patient population. *Am J Sports Med* 2009;37(7):1344-1350.
24. Gomoll AH, Probst C, Farr J, Cole BJ, Minas T: Use of a type I/III bilayer collagen membrane decreases reoperation rates for symptomatic hypertrophy after autologous chondrocyte implantation. *Am J Sports Med* 2009;37(suppl 1):20S-23S.
25. 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.
26. Pidoriario AJ, Weinstein RN, Buuck DA, Fulkerson JP: Correlation of patellar articular lesions with results from anteromedial tibial tubercle transfer. *Am J Sports Med* 1997;25(4):533-537.
27. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L: Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med* 1994;331(14):889-895.
28. Mandelbaum B, Browne JE, Fu F, et al: Treatment outcomes of autologous chondrocyte implantation for full-thickness articular cartilage defects of the trochlea. *Am J Sports Med* 2007;35(6):915-921.
29. Richmond J, Hunter D, Irrgang J, et al: Treatment of osteoarthritis of the knee (nonarthroplasty). *J Am Acad Orthop Surg* 2009;17(9):591-600.