Review Article

Cartilage Injury in the Knee: Assessment and Treatment Options

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Abstract

Cartilage injuries in the knee are common and can occur in isolation or in combination with limb malalignment, meniscus, ligament, and bone deficiencies. Each of these problems must be addressed to achieve a successful outcome for any cartilage restoration procedure. If nonsurgical management fails, surgical treatment is largely based on the size and location of the cartilage defect. Preservation of the patient's native cartilage is preferred if an osteochondral fragment can be salvaged. Chondroplasty and osteochondral autograft transfer are typically used to treat small (<2 cm^2) cartilage defects. Microfracture has not been shown to be superior to chondroplasty alone and has potential adverse effects, including cyst and intralesional osteophyte formation. Osteochondral allograft transfer and matrix-induced autologous chondrocyte implantation are often used for larger cartilage defects. Particulated juvenile allograft cartilage is another treatment option for cartilage lesions that has good to excellent short-term results but long-term outcomes are lacking.

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Focal cartilage defects result in disability that may be similar to osteoarthritis.[1] Symptoms include pain, swelling, stiffness, and locking or catching, all of which can limit patient activities. These lesions are often traumatic, as in the case of a patellar dislocation or concomitant anterior cruciate ligament (ACL) injury, but can also be caused by chronic repetitive overload. They may also be found incidentally on MRI or at the time of knee arthroscopy, which is an important distinction from a symptomatic defect. Cartilage lesions are problematic because hyaline articular cartilage has limited ability to regenerate in response to damage. Fibrocartilage filling may occur but many will progress with eventual development of arthritis.[2]

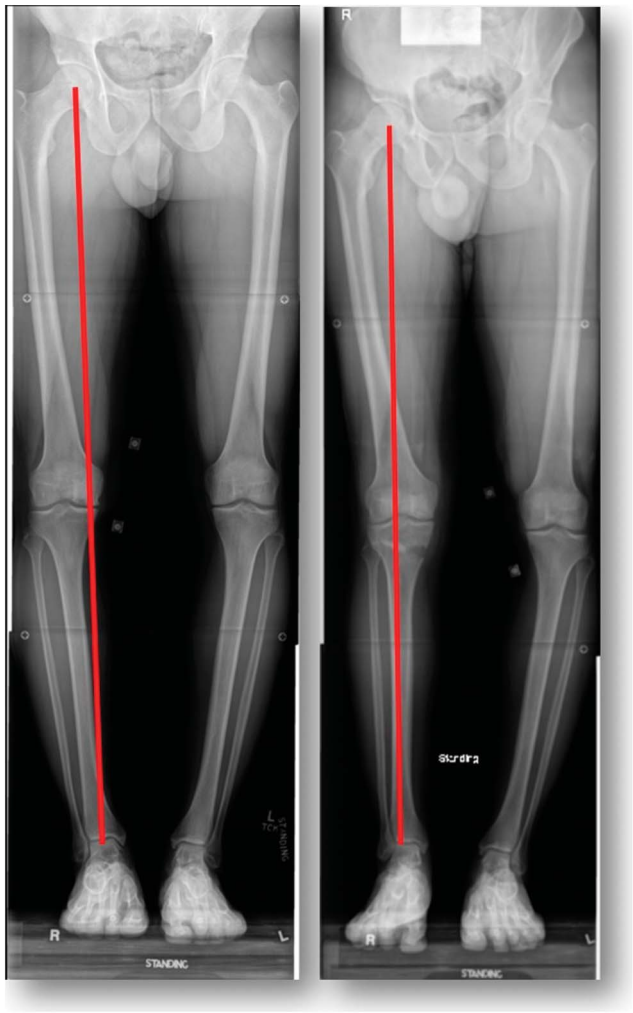
Radiographs are often unrevealing in a patient with acute knee pain after a cartilage injury. However, an effusion may be noted or a loose body may be present in the case of an osteochondral fracture. MRI is the preferred imaging modality to evaluate the depth, size, and location of a cartilage lesion and the subchondral bone.

There are several cartilage lesion categorization systems. The Outerbridge Classification described in 1961 is based on open or arthroscopic inspection of the cartilage surface. Outerbridge grade 0 describes normal cartilage, grade 1 represents cartilage softening to dynamic probing, grade 2 are partial thickness lesions less than 1.5 cm in diameter, grade 3 lesions are greater than 1.5 cm in diameter or have a full thickness fissure, and grade 4 lesions involve complete cartilage loss with exposed subchondral bone.[3] The International Cartilage Repair Society Classification is also based on

visual inspection of the cartilage and can guide management. Grade 0 describes normal intact cartilage. Grade 1 involves superficial cartilage lesions with softening, blistering, or fissures. Grade 2 cartilage lesions include fraying and fissures that are <50% of the cartilage depth. Grade 3 lesions describe cartilage loss that is >50% of the cartilage depth down to the calcified cartilage layer. Finally, grade 4 lesions are full-thickness cartilage lesions with exposure and involvement of the subchondral bone.

Small cartilage lesions, typically defined as less than 2 cm^2, are treated with a variety of options to include débridement, microfracture, fixation of unstable lesions, osteochondral autograft, or occasionally osteochondral allograft (OCA). Larger lesions are more typically managed with OCA or a cell-based option, such as matrix-induced autologous chondrocyte implantation (ACI). A patient with multiple large diffuse lesions throughout the knee should be carefully evaluated because this likely represents an osteoarthritis process. Some young, motivated patients can achieve good results, but older individuals may not be cartilage restoration candidates and may require arthroplasty.

Figure 1



Full length standing radiographs showing varus alignment (left image) and then correction to neutral alignment after medial proximal tibia opening wedge osteotomy (right image).

Concurrent Pathology

Once a cartilage lesion is identified, multiple variables must be evaluated when determining treatment options. The patient's clinical symptoms must correlate to physical examination and imaging findings to determine if the cartilage injury is the source of the patient's pain. In addition, a thorough ligamentous examination should be completed, assessing the cruciate and collateral ligaments and the patellofemoral joint. Ligament reconstruction surgery should take place before or concurrent to any cartilage procedures to obtain a successful outcome or else the cartilage treatment may be placed under excessive stress and fail. Lower extremity standing alignment should be assessed on examination for static or dynamic varus or valgus overload of the compartment and full length standing radiographs should be analyzed in all cases. A proximal tibial or distal femoral osteotomy should be considered if a 5° or greater mechanical axis deviation is present in the affected compartment[4] (Figure 1). Meniscus pathology should be carefully evaluated on MRI and arthroscopic examination. All attempts to preserve the meniscus through repair should be taken. The meniscus roots should always be examined and repaired if necessary because increased contact forces have been noted in the absence of an intact medial meniscus posterior root.[5] Patients with irreparable meniscal pathology and total/subtotal meniscectomy should be considered for a meniscal allograft transplantation.[6] Selective treatment of bipolar lesions can be considered.

Finally, patients with patellofemoral instability are evaluated with physical

examination, radiographs, MRI, and occasionally CT scans to identify lateral patellar tilt, patella alta, medial patellofemoral ligament incompetence, trochlear dysplasia, increased tibial tubercle-trochlear groove distance, and coronal plane or rotational malalignment. Surgical treatments are performed to address all anatomic factors that may place a patient at increased risk of recurrent dislocation or place excess stress on any cartilage procedures performed. Treatment options include but are not limited to tibial tubercle osteotomy, medial patellofemoral ligament reconstruction, and lateral retinacular lengthening at the time of patellofemoral cartilage surgery, if necessary. The purpose of these procedures is to decrease the risk of recurrent dislocations that may have led to the cartilage lesion and to decrease contact pressures on the cartilage by offloading the cartilage. For example, an anteromedialization tibial tubercle osteotomy can both decrease the risk of recurrent patellar dislocation by medializing the tubercle and offload distal pole of the patella or bipolar lesions by anteriorizing the tubercle.

Previous studies have demonstrated higher rates of cartilage restoration surgery failure when concurrent pathology is not addressed.[6] We recommend careful review of all imaging and clinical examination findings to prepare for all necessary procedures. Systematic arthroscopic or open surgical documentation of the cartilage lesions about size, confinement of the lesion, and condition of the cartilage surrounding the lesion and in the other knee compartments is of great importance to fully understand the potential background cause of the cartilage injury.

Débridement/ Chondroplasty

Arthroscopic débridement/chondroplasty is a technique in which a loose flap of cartilage that may be causing mechanical symptoms and/or effusions is débrided back to a stable edge. The goal of this technique is to help alleviate any mechanical symptoms and irritation, along with hopefully preventing the propagation of the cartilage lesion from any mechanical stress on the unstable flap.

Benefits of this procedure include the ability for immediate weight bearing with a shorter recovery period. This procedure is a relatively inexpensive treatment option that does not require notable preplanning or multiple stages as other cartilage restoration surgeries do. Studies have shown the procedure to be beneficial regarding pain, physical function, and quality of life when performed in the absence of concurrent pathology.[7] In a study of the National Football League athletes, 67% of patients were able to return to regular season National Football League play after chondroplasty.[8,9] Contrary to this, other studies have shown no benefit to debriding an unstable cartilage flap compared with observation alone in the setting of partial meniscectomy.[10] Unfortunately, the biggest limitation of surgical débridement is that it does not restore normal articular cartilage congruency because it only acts to treat mechanical symptoms from loose chondral flaps. Despite its limitations, this is a good first-line treatment option for patients with smaller cartilage lesions in the absence of concurrent pathology with the benefit of a short postoperative rehab period and certainly can be considered when the main symptom is mechanical in nature.

Fixation of Unstable Osteochondral Fragment or Loose Body

Unstable osteochondral fragments and loose bodies are commonly observed in the setting of patellofemoral instability or osteochondritis dissecans. If these fragments have viable cartilage and bone, typically 3 mm or greater on the progeny fragment, then consideration should be given to repair the fragment in the donor location. The presence of bone is ideal, but case series have shown that large chondral fragments without observable bone may do well with fixation in select cases in skeletally immature patients.[11] Skeletally mature and immature patients can benefit from fixation of unstable osteochondral fragments with healing rates not dependent on epiphyseal plate status.[12]

Fixation of these fragments is ideal because it uses native cartilage, can be performed in a single stage, and is relatively inexpensive. Unfortunately, a long-standing loose body may have poor quality cartilage, may resorb, or may hypertrophy, making it difficult to fix in the donor site.[13] We prefer to repair osteochondral fragments with viable bone and cartilage by thoroughly cleaning the fibrous tissue from the defect and fragment, adding an autologous bone graft if needed, and contouring and fixing the fragment.

The method of fixation varies and is largely dependent on fragment size and surgeon preference. Osteochondral defect progeny fragments can be fixed with headless compression screws, countersunk headed compression screws, or various bioabsorbable chondral darts and nails (Figure 2). The goal is to fix the osteochondral or chondral fragment to a healthy bleeding bone surface with good compression to allow for healing within the intra-articular environment of the knee.[11,14]

Microfracture

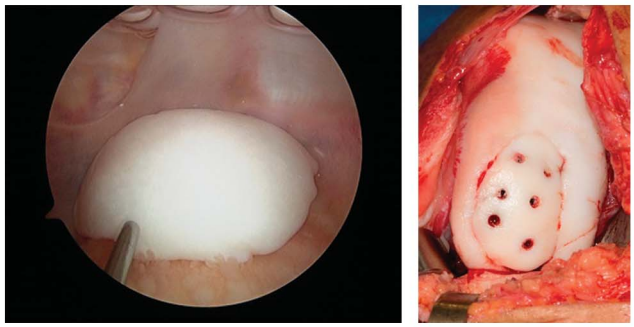
Microfracture is a technique that began in the 1980s but gained popularity in the early 2000s with the goal of treating articular cartilage defects in the knee by accessing bone marrow cells deep to the subchondral surface to heal full-thickness cartilage defects.[15] These marrow elements

contain growth factors that promote filling of the chondral defect with fibrocartilage. Microfracture is an option for small full-thickness cartilage lesions <2 cm^2 in size that are contained with a healthy rim of surrounding cartilage. Contraindications include global cartilage degeneration, inability to comply with weight bearing restrictions after the procedure, and a noncontained defect that a clot from the marrow contents will not form in.[15,16]

The procedure is performed using awls, picks, or drills to produce microfractures in the subchondral bone perpendicular to the surface and at least 3 to 4 mm apart from one another. It is important to have a stable rim of healthy intact cartilage around the chondral defect so that a clot can form in this area. In preparation of the site, studies have shown benefit to debriding the calcified cartilage layer so that only subchondral bone remains in the defect.[15,16] Blood and fat emanating from the site without a tourniquet inflated should be verified to assure that the subchondral surface was penetrated deep enough (Figure 3). This should be performed at the end of the procedure so that bleeding does not obstruct your view during arthroscopy and to allow clot formation at the cartilage defect site.

Historically, microfracture was most often used as an initial inexpensive and simple treatment. It was thought that there was no harm and that other cartilage restoration options would still be available to treat the cartilage lesion if microfracture failed. Some studies have shown satisfactory short-term results[17,18] but others demonstrate deterioration of function after 2 years, especially when compared with other treatment options for small cartilage defects.[19-21] Bone overgrowth occurs in more than 60% of patients, contributing to increased failure rates.[22] Animal studies also show altered subchondral architecture after microfracture with decreased bone mineral density.[23]

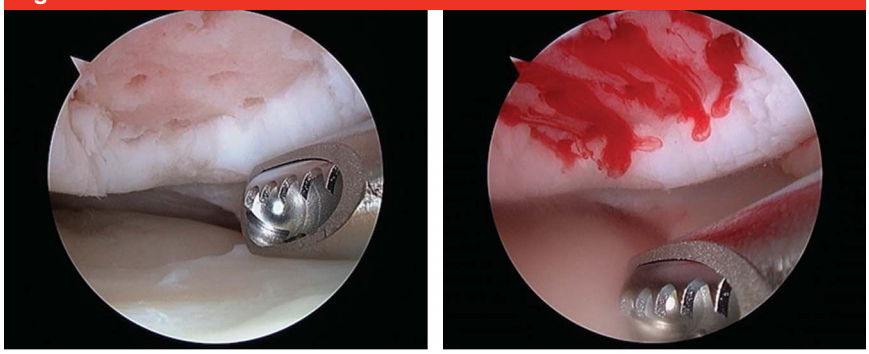
Figure 2



A B

Photographs showing the fixation of osteochondral loose body secondary to osteochondritis dissecans. **A**, Shows loose body retrieval during knee arthroscopy. Care is taken to not damage the cartilage while grasping fragment. **B**, After preparation of the donor site to good bleeding subchondral bone surface, fixation with multiple headless compression screws that are sunk below the cartilage surface is achieved.

Figure 3



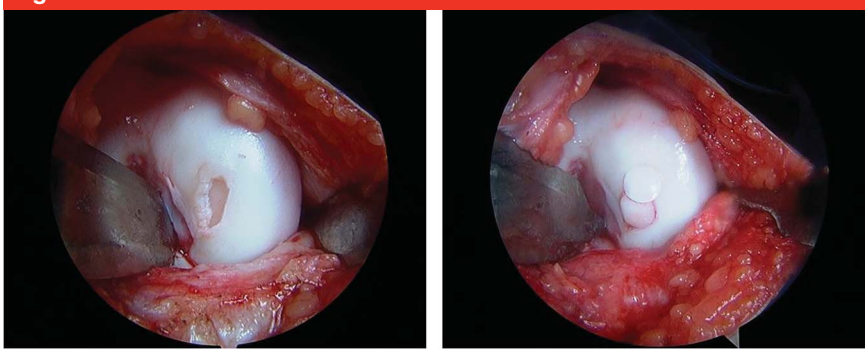
A B

Photographs showing the microfracture technique: (**A**) the cartilage lesion was débrided using a shaver and curets to a stable rim of healthy cartilage and removal of all calcified cartilage. Microfracture holes are seen approximately 3 to 4 mm apart from one another throughout the lesion. **B**, The tourniquet was let down, and blood is seen coming from all microfracture sites.

Osteochondral Autograft Transplant/Mosaicplasty

Osteochondral autograft transfer is a treatment best reserved for small osteochondral lesions in the knee that are <2 cm^2 in size. This treatment involves harvesting an osteochondral plug from a nonweight-bearing surface of the knee, typically the peripheral aspect of the medial or lateral trochlea or intercondylar notch and then transferring this to a weight-bearing chondral lesion. The plugs harvested are typically 6 to 10 mm in size, and numerous plugs can be used in a mosaic format if needed for larger defects (Figure 4). The plug itself offers a hyaline cartilage surface with underlying subchondral bone which assists in the healing process. If gaps are present, then fibrocartilage fills in

Figure 4



A B

Photographs showing the osteochondral autograft transfer with two plugs: (**A**) the small contained cartilage lesion is seen. **B**, Two osteochondral autograft plus are fitted in to the defect in a snowman configuration.

the surrounding defects around the osteochondral autograft plug and native surrounding cartilage. The procedure has been described both with an open arthrotomy and arthroscopically. The grafts are typically press fit into the lesion, and no hardware is necessary if stable fixation is able to be achieved.[24] Because of donor site morbidity, this treatment is typically limited to lesions less than 2 cm^2 in size. In our opinion, the ideal indication would be a transfer of one or two 8-mm osteochondral autograft plugs. Therefore, in addition to lesion size, the geometry must also be favorable with a narrow, linear lesion. We find that few lesions of this size are symptomatic, and therefore, there are limited indications for this technique.

Advantages include a single stage procedure, lower cost compared with an allograft, and the ability to treat lesions with subchondral bone involvement. Utilization of the patient's native cartilage and living bone should theoretically improve healing potential. Osteochondral autologous transplantation (OAT) results in improved subjective scores, higher rates of return to sport in athletes, 89% versus 51% in OAT compared with microfracture,[25] and lower failure rates at long-term follow-up when compared with microfracture.[9,25-27] At the 3-year follow-up, 86% of OAT patients had continuation of sport compared with just 27% of microfracture patients in an athletic cohort. These results further decreased at the 10-year follow-up, with 34% and 17% of the OAT and microfracture patients, respectively, continuing to participate in sporting activities.[25]

A limitation of this technique is that it can be difficult to contour match the donor cartilage to the lesion to create a congruent surface. In addition, larger lesions are more difficult to treat because they necessitate a mosaic construct, and there is concern for donor site morbidity.

Osteochondral Allograft Transplant

OCAs have shown to be successful for a variety of cartilage lesions. In particular, these grafts are useful in young healthy patients with large and deeper osteochondral lesions >2 cm^2 in size. These grafts may also be used for those with less-contained cartilage defects, for those with involvement of the underlying subchondral bone, and offer an elegant solution in revision settings. OCAs are not ideal for obese patients or those who use tobacco or corticosteroids because research has shown higher failure rates in these patients, as well as those with inflammatory arthritis.[28,29]

Historically, fresh OCAs were implanted within 24 to 48 hours; but, with more rigorous regulations now in place, these grafts are not typically implanted until a minimum of 14 days postharvest to allow for final aerobic cultures to be negative. Graft chondrocyte viability has been shown to be directly proportional to time since harvest; so, once the graft is released to the surgeon, the surgery is typically scheduled within 1 to 2 weeks.[30] OCAs have viable cartilage but require creeping substitution for bone integration with the host bone and therefore require a period of nonweight bearing or toe-touch weight-bearing after surgery if on a weight-bearing surface to allow for healing.[29]

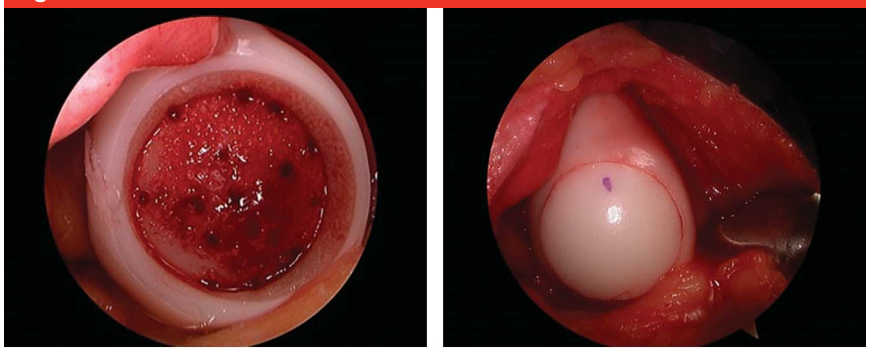
Surgical technique involves debriding the cartilage lesion and subchondral bone to a stable healthy rim. OCA-specific instrumentation can then be used for the recipient and donor site to create a press-fit fixation of the allograft (Figure 5). Contouring of uncontained lesions must be performed by the surgeon to allow for appropriate size and depth of the graft. These uncontained lesions may require compression screw fixation to stabilize the graft.

OCAs have shown promising outcomes in past studies when used for the correct patient cohort and when concurrent pathology is also corrected. Generally, studies report approximately 82% success rate after the surgery with most failures occurring on average approximately 42 months after surgery.[29,31] More complex lesions that require multiple grafts in a snowman configuration have higher failure and revision surgery rates as compared to a single graft, but overall, patients experience notable clinical improvement.[32] OCAs also provide mature hyaline cartilage that is an immediate functional surface for

rehabilitation and loading, unlike cell-based options that require maturation over time. Limitations include limited availability, waiting time, and the high cost of a fresh OCA. Recent efforts have been made to release the grafts available as soon as testing is completed.

Fresh OCA precut plugs up to 16 mm in diameter are also now available. These plugs are limited by their shelf life but do provide an option for osteochondral lesions that are considered too large for osteochondral autograft because of potential donor site morbidity. Further research is needed to determine the long-term outcomes of these grafts.[33]

Figure 5



A B

Photographs of the osteochondral allograft (OCA) transfer (**A**) shows a lesion that has been cut and reamed with a clean rim of healthy cartilage and bleeding subchondral bone. **B**, Shows an OCA that has been press fit to the native femoral condyle. The small mark on the cartilage is used to denote orientation of the graft from the allograft donor to the recipient site.

Matrix-Induced Autologous Chondrocyte Implantation

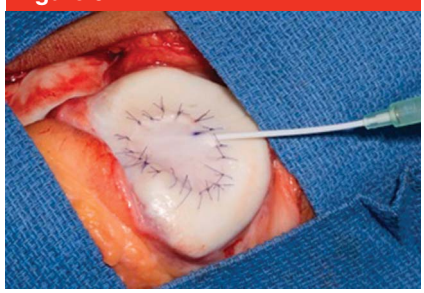
Matrix-induced autologous cultured chondrocytes implantation (MACI) was approved by the FDA in 2017 in the United States and is currently the only FDA-approved cell-based cartilage treatment option. MACI has evolved from the excellent long-term track record of ACI but fulfills the need for a more efficient technique. Previously, first-generation ACI used an autologous periosteal membrane, with the second generation using a bioabsorbable collage membrane sewn into place, under which cultured chondrocytes were injected (Figure 6). MACI uses a membrane that acts as a cell carrier to more evenly distribute the cells with a density of 500,000 to 1,000,000 cells per cm^2 and is easier to implant.[34,35]

MACI is typically used in patients with an articular cartilage lesion greater than 2 cm^2 in size who have failed nonsurgical treatment. It has the advantage of being a form-fitting membrane that can fit into a variety of different size and shape lesions.

MACI is a two-stage procedure, with the first stage involving cartilage biopsy that is then sent to a laboratory for culturing of the chondrocytes on a collagen membrane. The biopsy most typically is harvested from either the intercondylar notch, the proximal aspect of the medial or lateral femoral condyle, or from the rim of the lesion. During the second stage of the procedure, the defect is débrided to stable edges with vertical walls. Any remaining cartilage is curetted down to the level of the calcified cartilage to create a contained defect. The graft is then prepared to the same size and shape. This can be performed using preshaped cutting tools or can be performed free hand.[34,36] During implantation, fibrin glue is applied within the defect bed, and the membrane is then gently compressed down against the defect. After securing the membrane, an additional thin even layer of fibrin glue is applied over the membrane and allowed to cure. The final result should be a stable membrane that evenly fills the defect (Figure 7).

The results after MACI have been promising and demonstrate notable clinical improvement when compared with microfracture in randomized prospective studies at the 5-year follow-up.[35] Studies have shown an estimated 9% to 10% revision surgery rate in MACI/ACI patients with symptomatic overgrowth of the cartilage is a potential source of revision surgery.[34,35] The fact that MACI requires two stages and being expensive are the greatest limitations to this procedure.[37]

Figure 6

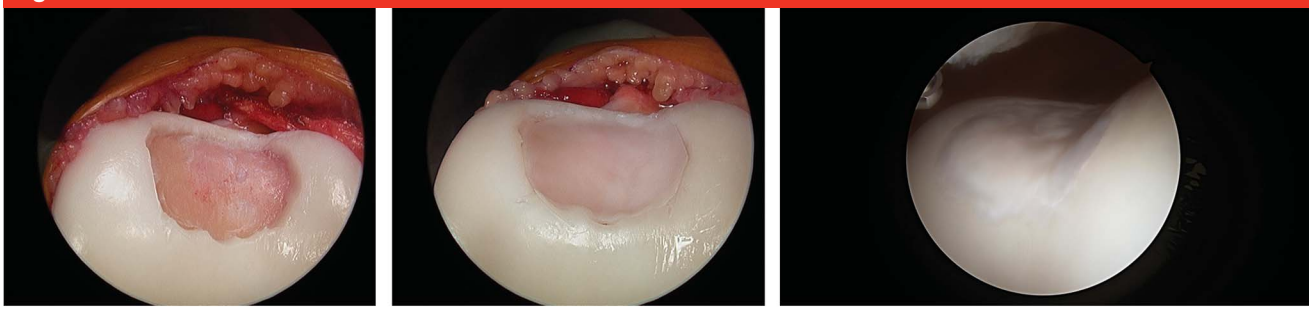


Photographs of the autologous chondrocyte implantation with a collagen membrane sewn in place with injection of autologous cultured chondrocytes under the membrane.

Particulated Juvenile Allograft Cartilage

Particulated juvenile allograft cartilage (PJAC) involves implantation of immature chondrocytes. Juvenile cartilage is thought to have improved chondrogenic activity as compared to adult cartilage and therefore makes it a

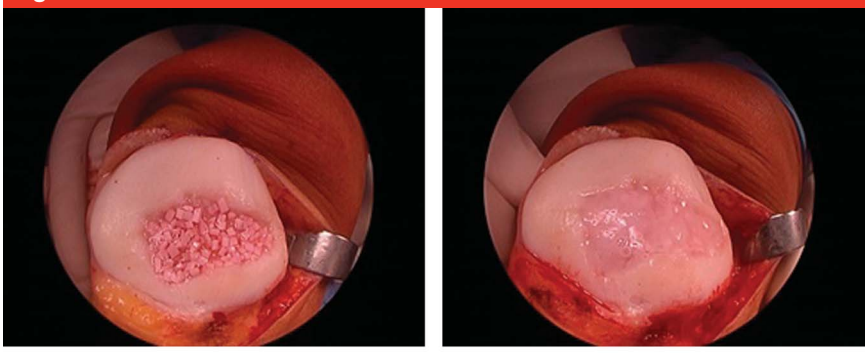
Figure 7



A B C

Photographs showing the matrix-induced autologous cultured chondrocytes implantation. **A**, Demonstrates a >2 cm^2 trochlear lesion after it has been cleared of fibrous tissue and stable walls now surround the defect. **B**, A size- and shape-matched graft has been secured with fibrin glue. **C**, Second look procedure demonstrates the graft has nicely filled in the defect with stable rims.

Figure 8



A B

Photographs showing the particulated juvenile allograft cartilage. **A**, Particulated juvenile allograft cartilage evenly filling the defect on a bed of fibril glue. **B**, An additional layer of fibril glue is applied over the cartilage sealing it in place.

suitable option for cartilage restoration procedures.[38] This treatment is most often used for large >2 cm^2 grade three or four patellar lesions that have failed nonsurgical treatment. Concurrent pathology such as patellar instability should also be addressed at the time of surgery.

Similar to MACI/ACI, this procedure involves débridement of the lesion to a stable rim and to healthy subchondral bone. Fibrin glue is then laid in the lesion, followed by an even distribution of PJAC and a subsequent layer of fibrin glue (Figure 8). PJAC provides the benefit of ease of contour matching and therefore is a suitable option for patellar chondral lesions. The main advantage of PJAC is that it is a one stage procedure, unlike the two stage surgery required for MACI/ACI.[38,39]

Studies have shown symptomatic improvement in patients with patellar chondral lesions at the short-term follow-up after this procedure, but no randomized controlled studies have been performed comparing the outcomes of PJAC with other cartilage procedures in the knee. A common complication similar to MACI/ACI is graft hypertrophy.[38,39] Most research on PJAC has been focused on talus lesions in the ankle, and therefore, further studies are needed comparing PJAC with other common cartilage restoration procedures in the knee.

Postoperative Rehabilitation

Surgeons have varying protocols regarding postoperative rehabilitation after cartilage restoration procedures. Range of motion is usually encouraged within the first week after all techniques to promote cartilage healing and prevent stiffness. Weight bearing is more heterogeneous and dependent on the procedure type and location along with surgeon preference. In general, weight bearing as tolerated is allowed immediately after chondroplasty. Full weight bearing in extension is typically tolerated within a week or two of a patellar cartilage procedure. Microfracture, MACI, and PJAC do not provide an immediate functional mature cartilage surface for loading require 6 weeks of protected weight bearing, followed by a progression to full weight bearing. Although there are no universal rehabilitation protocols, osteochondral autograft and allograft procedures generally allow for a more rapid return to full weight bearing. Some surgeons allow full weight

bearing in the first 6 weeks, and most surgeons allow the return to full weight bearing by 6 weeks postoperatively.[40]

Summary

Cartilage injuries in the knee are a challenging problem for both patients and physicians. Treatment depends on individual patient characteristics and the lesion location, size, and depth. All concurrent pathology must be addressed, including ligament instability, meniscus deficiency, and limb malalignment for cartilage restoration procedures to be successful. The first goal of any cartilage procedure is to preserve the patient's own cartilage if possible. Patients with small symptomatic lesions <2 cm^2 who have failed nonsurgical management are treated with chondroplasty or osteochondral autograft transfer. Larger defects require OCA transplantation, matrix-induced ACI, or PJAC. Continued advances in cartilage restoration procedures will positively provide single stage and cost-effective treatment options for patients in the future.

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