A Review Paper

Cartilage Restoration in the Patellofemoral Joint

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Abstract

Although patellofemoral (PF) chondral lesions are common, the presence of a cartilage lesion does not implicate a chondral lesion as the sole source of pain. As attributing PF pain to a chondral lesion is "diagnosis by exclusion," thorough assessment of all potential structural and nonstructural sources of pain is the key to proper management. Commonly, multiple factors contribute to a patient's symptoms. Each comorbidity must be identified and addressed, and the cartilage lesion treatment determined.

Comprehensive preoperative assessment is essential and should include a thorough "core-to-floor" physical examination. Treatment of symptomatic chondral lesions in the PF joint requires specific technical and postoperative management, which differs significantly from management involving the tibiofemoral joint. Attending to all these details makes the outcomes of PF cartilage treatment reproducible. These outcomes may rival those of chondral repair in the tibiofemoral joint.

Patellofemoral (PF) pain is often a component of more general anterior knee pain. One source of PF pain is chondral lesions. As these lesions are commonly seen on magnetic resonance imaging (MRI) and during arthroscopy, it is necessary to differentiate incidental and symptomatic lesions.[1] In addition, the correlation between symptoms and lesion presence and severity is poor.

PF pain is multifactorial (structural lesions, malalignment, deconditioning, muscle imbalance and overuse) and can coexist with other lesions in the knee (ligament tears, meniscal injuries, and cartilage lesions in other compartments). Therefore, careful evaluation is key in attributing knee pain to PF cartilage lesions—that is, in making a "diagnosis by exclusion."

From the start, it must be appreciated that the vast majority of patients will not require surgery, and many who require surgery for pain will not require cartilage restoration. One key to success with PF patients is a good working relationship with an experienced physical therapist.

Etiology

The primary causes of PF cartilage lesions are patellar instability, chronic maltracking without instability, direct trauma, repetitive microtrauma, and idiopathic.

Patellar Instability

Patients with patellar instability often present with underlying anatomical risk factors (eg, trochlear dysplasia, increased Q-angle/tibial tubercle-trochlear groove [TT-TG] distance, patella alta, and unbalanced medial and lateral soft tissues[2]). These factors should be addressed before surgery.

Patellar instability can cause cartilage damage during the dislocation event or by chronic subluxation. Cartilage becomes damaged in up to 96% of patellar dislocations.[3] Most commonly, the

Take-Home Points

- Careful evaluation is key in attributing knee pain to patellofemoral cartilage lesions—that is, in making a "diagnosis by exclusion."
- Initial treatment is nonoperative management focused on weight loss and extensive "core-to-floor" rehabilitation.
- Optimization of anatomy and biomechanics is crucial.
- Factors important in surgical decision-making include defect location and size, subchondral bone status, unipolar vs bipolar lesions, and previous cartilage procedure.
- The most commonly used surgical procedures—autologous chondrocyte implantation, osteochondral autograft transfer, and osteochondral allograft—have demonstrated improved intermediate-term outcomes.

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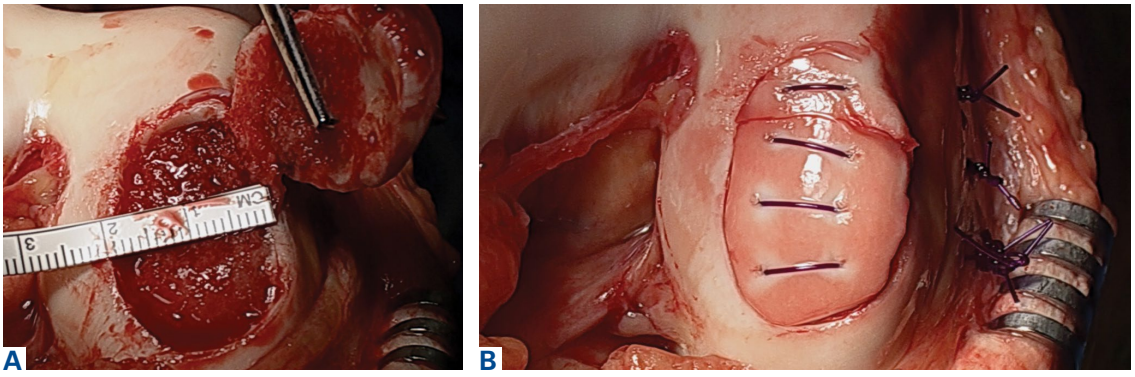

Figure 1. (A) Lateral femoral condyle osteochondral lesion, caused by patellar dislocation, is predominantly cartilaginous. (B) Fixation of predominantly cartilaginous fragment with No. 1 monofilament resorbable transosseous sutures.

damage consists of fissuring and/or fibrillation, but chondral and osteochondral fractures can occur as well. During dislocation, the medial patella strikes the lateral aspect of the femur, and, as the knee collapses into flexion, the lateral aspect of the proximal lateral femoral condyle (weight-bearing area) can sustain damage. In the patella, typically the injury is distal-medial (occasionally crossing the median ridge). A shear lesion may involve the chondral surface or be osteochondral (**Figure 1A**). In an osteochondral lesion, the area of cartilage damage is often larger than the bony fragment indicates (Figure 1A), and even small fractures visible on radiographs can portend extensive cartilage damage. In addition, isolated cartilage flaps can occur; if suspected, they should be assessed with MRI. The extent of cartilage damage is related to the magnitude of energy required to cause the dislocation and/or to the frequency of events. In more normal anatomy, more energy is required to provoke a dislocation, and damage to articular cartilage is greater. In recurrent patellar dislocation, each event can cause additional injury, and the size of the lesion tends to increase with the number of dislocations.[4] Patellar dislocation can result in chronic patellar subluxation, or dislocations that often lead to recurrent or chronic patellar instability. With recurrent instability, the medial patellar facet becomes damaged as it displaces out of the trochlea during subluxation and dislocation events. With lateral patellar maltracking, the contact area is reduced. With overall similar PF forces, a smaller contact area results in increased point loading, thus increasing stress and promoting cartilage wear.

Chronic Maltracking Without Instability

Chronic maltracking is usually related to anatomical abnormalities, which include the same factors that can cause patellar instability. A common combination is trochlear dysplasia, increased TT-TG or TT-posterior cruciate ligament distance, and lateral soft-tissue contracture. These are often seen in PF joints that progress to lateral PF arthritis. As lateral PF arthritis progresses, lateral soft-tissue contracture worsens, compounding symptoms of laterally based pain. With respect to cartilage repair, these joints can be treated if recognized early; however, once osteoarthritis is fully established in the joint, facetectomy or PF replacement may be necessary.

Direct Trauma

With the knee in flexion during a direct trauma over the patella (eg, fall or dashboard trauma), all zones of cartilage and subchondral bone in both patella and trochlea can be injured, leading to macrostructural damage, chondral/osteochondral fracture, or, with a subcritical force, microstructural damage and chondrocyte death, subsequently causing cartilage degeneration (cartilage may look normal initially; the matrix takes months to years to deteriorate). Direct trauma usually occurs with the knee flexed. Therefore, these lesions typically are located in the distal trochlea and superior pole of the patella.

Repetitive Microtrauma

Minor injuries, which by themselves do not immediately cause apparent chondral or osteochondral fractures, may eventually exceed the capacity of natural cartilage homeostasis and result in repetitive microtrauma. Common causes are repeated jumping (as in basketball and volleyball) and prolonged flexed-knee position (eg, what a baseball catcher experiences), which may also be associated with other lesions caused by extensor apparatus overload (eg, quadriceps tendon or patellar tendon tendinitis, and fat pad impingement syndrome).

Idiopathic

In a subset of patients with osteochondritis dissecans, the patella is the lesion site. In another subset, idiopathic lesions may be related to a genetic predisposition to osteoarthritis and may not be restricted to the PF joint. In some cases, the PF joint is the first compartment to degenerate and is the most symptomatic in a setting of truly tricompartmental disease. In these cases, treating only the PF lesion can result in functional failure, owing to disease progression in other compartments. Even mild disease in other compartments should be carefully evaluated.

History and Physical Examination

Patients often report a history of anterior knee pain that worsens with stair use, prolonged sitting, and flexed-knee activities (eg, squatting). Compared with pain alone, swelling, though not specific to cartilage disease, is more suspicious for a cartilage etiology. Identifying the cartilage defect as the sole source of pain is particularly difficult in patients with recurrent patellar instability. In these patients, pain and swelling, even between instability episodes, suggest that cartilage damage is at least a component of the symptomology.

Important diagnostic components of physical examination are gait analysis, tibiofemoral alignment, and patellar alignment in all 3 planes, both static and functional. Patella-specific measurements include medial-lateral position and quadrants of excursion, lateral tilt, and patella alta, as well as J-sign and subluxation with quadriceps contraction in extension.

It is also important to document effusion; crepitus; active and passive range of motion (spine, hips, knees); site of pain or tenderness to palpation (medial, lateral, distal, retropatellar) and whether it matches the complaints and the location of the cartilage lesion; results of the grind test (placing downward force on the patella during flexion and extension) and whether they match the flexion angle of the tenderness and the flexion angle in which the cartilage lesion has increased PF contact; ligamentous and soft-tissue stability or imbalance (tibiofemoral and patellar; apprehension test, glide test, tilt test); and muscle strength, flexibility, and atrophy of the core (abdomen, dorsal and hip muscles) and lower extremities (quadriceps, hamstrings, gastrocnemius).

Imaging

Imaging should be used to evaluate both PF alignment and the cartilage lesions. For alignment, standard radiographs (weight-bearing knee sequence and axial view; full limb length when needed), computed tomography, and MRI can be used.

Meaningful evaluation requires MRI with cartilage-specific sequences, including standard spin-echo (SE) and gradient-recalled echo (GRE), fast SE, and, for cartilage morphology, T2-weighted fat suppression (FS) and 3-dimensional SE and GRE.[5] For evaluation of cartilage function and metabolism, the collagen network, and proteoglycan content in the knee cartilage matrix, consideration should be given to compositional assessment techniques, such as T2 mapping, delayed gadolinium-enhanced MRI of cartilage, T1ρ imaging, sodium imaging, and diffusion-weighted sequences.[5] Use of the latter functional sequences is still debatable, and these sequences are not widely available.

Treatment

In general, the initial approach is nonoperative management focused on weight loss and extensive core-to-floor rehabilitation, unless surgery is specifically indicated (eg, for loose body removal or osteochondral fracture reattachment). Rehabilitation focuses on achieving adequate range of motion of the spine, hips, and knees along with muscle strength and flexibility of the core (abdomen, dorsal and hip muscles) and lower limbs (quadriceps, hamstrings, gastrocnemius). Rehabilitation is not defined by time but rather by development of an optimized soft-tissue envelope that decreases joint reactive forces. The full process can take 6 to 9 months, but there should be some improvement by 3 months.

Corticosteroid, hyaluronic acid,[6] or platelet-rich plasma[7] injections can provide temporary relief and facilitate rehabilitation in the setting of pain inhibition. As stand-alone treatment, injections are more suitable for more diffuse degenerative lesions in older and low-demand patients than for focal traumatic lesions in young and high-demand patients.

Surgery is indicated for full-thickness or nearly full-thickness lesions (International Cartilage Repair Society grade 3a or higher) >1 cm^2 after failed conservative treatment.

Optimization of anatomy and biomechanics is crucial, as persistent abnormalities lead to high rates of failure of cartilage procedures, and correction of those factors results in outcomes similar to those of patients without such abnormal anatomy.[8] The procedures most commonly used to improve

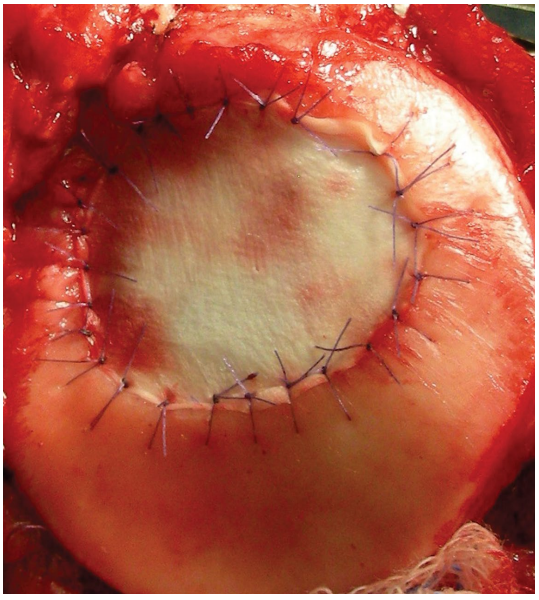

Figure 2. Patella autologous chondrocyte implantation with a 6.0 resorbable braided suture.

patellar tracking or unloading in the PF compartment are lateral retinacular lengthening and TT transfer: medialization and/or distalization for correction of malalignment, and straight anteriorization or anteromedialization for unloading. These procedures can improve symptoms and function in lateral and distal patellar and trochlear lesions even without the addition of a cartilage restoration procedure.

Factors that are important in surgical decision-making include defect location and size, subchondral bone status, unipolar vs bipolar lesions, and previous cartilage procedure.

- *Location*. The shapes of the patella and trochlea vary much more than the shapes of the condyles and plateaus. This variability complicates morphology matching, particularly with involvement of the central TG and median patellar ridge. Therefore, focal contained lesions of the patella and trochlea may be more technically amenable to cell therapy techniques than to osteochondral procedures, which require contour matching between donor and recipient.
- *Size*. Although small lesions in the femoral condyles can be considered for microfracture (MFx) or osteochondral autograft transfer (OAT), MFx is less suitable because of poor results in the PF joint, and OAT because of donor-site morbidity in the trochlea.
- *Subchondral bone status*. When subchondral bone is compromised, such as with bone loss, cysts, or significant bone edema, the entire osteochondral unit should be treated. Here, OAT and osteochondral allograft (OCA) are the preferred treatments, depending on lesion size.
- *Unipolar vs bipolar lesions*. Compared with unipolar lesions, bipolar lesions tend to have worse outcomes. Therefore, an associated unloading procedure (TT osteotomy) should be given special consideration. Autologous chondrocyte implantation (ACI) appears to have better outcomes than OCA for bipolar PF lesions.[9,10]
- *Previous surgery*. Although a failed cartilage procedure can negatively affect ACI outcomes, particularly in the presence of intralesional osteophytes,[11] it does not affect OCA outcomes.[12] Therefore, after previous MFx, OCA instead of ACI may be considered.

Fragment Fixation

Viable fragments from traumatic lesions (direct trauma or patellar dislocation) or osteochondritis dissecans should be repaired if possible, particularly in young patients. In a fragment that contains a substantial amount of bone, compression screws provide stable fixation. More recently, it has been recognized that fixation of predominantly cartilaginous fragments can be successful[13] (**Figure 1B**). Débridement of soft tissue in the lesion bed and on the fragment is important in facilitating healing, as is removal of sclerotic bone.

MFx

Although MFx can have good outcomes in small contained femoral condyle lesions, in the PF joint treatment has been more challenging, and clinical outcomes have been poor (increased subchondral edema, increased effusion).[14] In addition, deterioration becomes significant after 36 months. Therefore, MFx should be restricted to small (<2 cm^2), well-contained trochlear defects, particularly in low-demand patients.

ACI and Matrix-Induced ACI

As stated, ACI (**Figure 2**) is suitable for PF joints because it intrinsically respects the complex anatomy. Multiple case series with midterm and long-term follow-up have found improved outcomes for patella and trochlea.[8,15] With careful assessment and correction of malalignment, outcomes are similar to those of patients with normal anatomy.[8] Results tend to be better for unipolar lesions than for bipolar lesions.[15] TT osteotomy is a useful adjunct in correcting malalignment and unloading the PF com-

partment, even more so in the bipolar lesion setting. Previous procedures that violate the subchondral bone increase the risk of failure of subsequent ACI 3- to 7-fold, particularly in the presence of persistent subchondral abnormalities, such as intralesional osteophytes, cysts, and significant edema.[11]

OAT

As mentioned, donor-site morbidity may compromise final outcomes of harvest and implantation in the PF joint. Nonetheless, in carefully selected patients with small lesions that are limited to 1 facet (not including the patellar ridge or the TG) and that require only 1 plug (**Figure 3**), OAT can have good clinical results.[16]

OCA

Two techniques can be used with OCA in the PF joint. The dowel technique, in which circular plugs are implanted, is predominantly used for defects that do not cross the midline (those located in their entirety on the medial or lateral aspect of the patella or trochlea). Central defects, which can be treated with the dowel technique as well, are technically more challenging to match perfectly, because of the complex geometry of the median ridge and the TG (**Figure 4**). The shell technique is an alternative that can be used to treat very large defects. The chondral defect area and subchondral bone are removed with an oscillating saw, using the same plane as for patellar resurfacing (total knee arthroplasty or PF arthroplasty). A matching graft is created with a similar cut, made freehand.

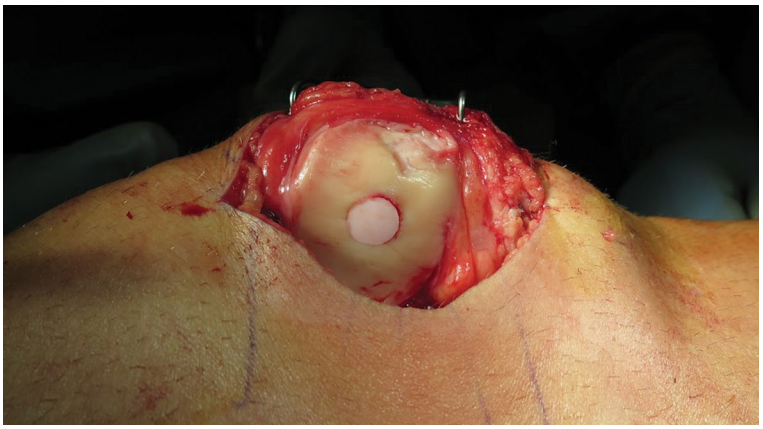

Figure 3. Osteochondral autograft transfer in the medial patellar facet.

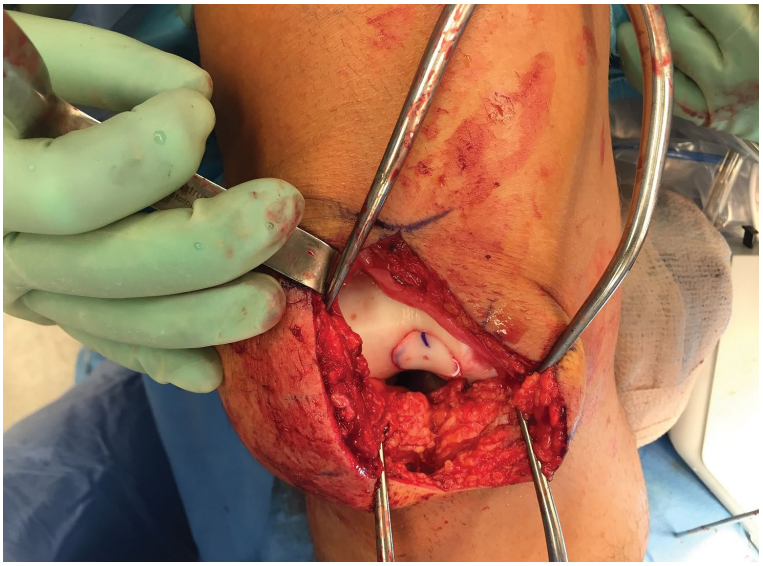

Figure 4. Allograft by the dowel technique in the trochlear groove maintains anatomy of the sulcus.

Experimental and Emerging Technologies

Biocartilage

Biocartilage, a dehydrated, micronized allogeneic cartilage scaffold implanted with platelet-rich plasma and fibrin glue added over a contained MFx-treated defect, can be used in the patella and trochlea and has the same indications as MFx (small lesions, contained lesions). There are limited clinical studies of short- or long-term outcomes.

Fresh and Viable OCA

Fresh OCA (ProChondrix; AlloSource) and viable/cryopreserved OCA (Cartiform; Arthrex) are thin osteochondral scaffolds that contain viable chondrocytes and growth factors. They can be implanted alone or used with MFx, and are indicated for lesions measuring 1 cm^2 to 3 cm^2. Aside from a case report,[17] there are no clinical studies on outcomes.

Bone Marrow Aspirate Concentrate Implantation

Bone marrow aspirate concentrate from centrifuged iliac crest–harvested aspirate containing mesenchymal stem cells with chondrogenic potential is applied under a synthetic scaffold. Indications are the same as for ACI. Medium-term follow-up studies in the PF joint have shown good results, similar to those obtained with matrix-induced ACI.[18]

Particulated Juvenile Allograft Cartilage

Particulated juvenile allograft cartilage (DeNovo NT Graft; Zimmer Biomet) is minced cartilage allograft (from juvenile donors) that has been cut into cubes (~1 mm^3). Indications are for patellar and trochlear lesions 1 cm^2 to 6 cm^2. For both the trochlea

and the patella, short-term outcomes have been good.[19,20]

Rehabilitation After Surgery

Isolated PF cartilage restoration generally does not require prolonged weight-bearing restrictions, and ambulation with the knee locked in full extension is permitted as tolerated. Concurrent TT osteotomy, however, requires protection with 4 to 6 weeks of toe-touch weight-bearing to minimize the risk of tibial fracture.

Conclusion

Comprehensive preoperative assessment is essential and should include a thorough core-to-floor physical examination as well as PF-specific imaging. Treatment of symptomatic chondral lesions in the PF joint requires specific technical and postoperative management, which differs significantly from management involving the condyles. Attending to all these details makes the outcomes of PF cartilage treatment reproducible. These outcomes may rival those of condylar treatment.

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