Considerations in Evaluating Treatment Options for Patellofemoral Cartilage Pathology

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Abstract: Patellofemoral (PF) pain, a subset of anterior knee pain, presents a particularly challenging diagnosis due to the multifactorial etiology. Within this group, assigning the patient's symptoms to a patellofemoral cartilage lesion is indirect; that is, a diagnosis by exclusion as hyaline cartilage is aneural. In addition, these PF compartment lesions are often in conjunction with various comorbidities, for example, malalignment and/or instability. In light of these factors and the high shear and compression stresses at the PF compartment, patellar and trochlear chondral lesions require unique treatment considerations from the tibiofemoral compartments. A thorough understanding of the various cartilage restoration techniques available is necessary to select the best option for the individual patient/knee/lesion noting that there is overlap of techniques' applications. In addition, failure to address and correct associated comorbidities may jeopardize the outcome of any cartilage restoration procedure. That is, the key to achieving optimal outcomes with PF cartilage restoration is to select the best cartilage treatment for the particular setting and to concomitantly optimize the PF biomechanical environment and stability.

Key Words: patellofemoral, cartilage, patella, trochlea, chondral defect

(*Sports Med Arthrosc Rev* 2016;24:92–97)

Anterior knee pain is a common complaint of patients seeking treatment by both primary care physicians and orthopedic surgeons. One subset of anterior knee pain is patellofemoral (PF) pain: in itself a challenging diagnosis because of the multifactorial etiology of the pain. For years the term patellofemoral chondromalacia was used to describe this disorder. This is incorrect as articular cartilage is aneural and many patients with anterior knee pain do not have cartilage defects and many with chondrosis have no pain. However, there is a subset of patients with PF pain that have chondral defects that through a "diagnosis by exclusion" can be assigned to the group of symptomatic PF cartilage lesions. Within the group, there will be multiple PF comorbidities/cofactors such as malalignment, tilt, and/or ligament insufficiency.

In patients with high-grade articular cartilage lesions of the knee (ICRS $\geq$ 3a) the patella is the second most common location after the medial femoral condyle.[1–3] Patellar and trochlear lesions present unique treatment considerations in light of high shear and compression stresses across the joint, and the multiple cofactors noted above. Chondral lesions of the PF joint may arise from both macrotrauma and microtrauma. In the case of macrotrauma, it may be a direct impact injury or occur with patellar instability events. Microtrauma causes chondrosis over time typically associated with abnormal joint loading (eg, chronic patellar subluxation causing lateral PF wear or repetitive high loading with sports causing central trochlear injury). The true prevalence of PF cartilage defects is controversial because it is unknown how many lesions are symptomatic as evidenced by the number of "asymptomatic PF lesions" noted when NBA players are routinely scanned with magnetic resonance imaging (MRI).[4]

HISTORY

Patients will often complain of anterior knee pain that may worsen with stair descent greater than ascension, squatting activities, or other high PF loading activities. Large chondral defects may cause clicking, popping, or swelling. As the articular cartilage is aneural, this pain is by definition secondary. The most common contributor to focal pain is probably subchondral bone overload, whereas more global pain is likely from synovial or capsular inflammation. Certainly, neural pain or even referred pain (eg, hip), must be excluded as well as complex regional pain syndrome and the pain of debilitation. In a subset of patients, they will have a history of a traumatic patellar dislocation or subluxation event. Note that chondral defects of the patella may be seen in as many as 95% of patients after a patellar dislocation event—though most do not warrant restoration considerations.[5]

PHYSICAL EXAM

The exam should be systematic and an extension of a standard knee examination. During gait and stance the limb alignment is quantified. Frequently these patients will have increased femoral anteversion (squinting of the patella) and valgus malalignment. The historical quadriceps angle (*Q*-angle) may be evaluated in both full extension and 30 degrees of flexion. The *Q*-angle is the angle between the lines connecting the center of the patella to the anterosuperior iliac spine proximally and the tibial tubercle distally. Although still commonly measured (average *Q*-angles in asymptomatic patients are 14 degrees in males and 17 degrees in females[6]), an excellent review article by Post[7] included a review of the *Q*-angle and reported such variability in the angle that no surgical decision can be based on this measurement alone.

Functional testing includes stepping up and down, forward and backward to assess limb control—especially loss of pelvic stability and hip abductors allowing dynamic internal rotation and valgus positioning.

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Disclosure: J.F. reports personal fees and nonfinancial support from Zimmer Biomet, outside the submitted work. Full list of disclosures is up to date at aaos.org. G.J.P. declares no conflict of interest.

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Sitting allows assessment of patellar tracking through range of motion with attention to arcs of marked crepitus. Terminal "J" tracking may suggest trochlear dysplasia or marked alta that allows the patella to exit the trochlear containment.

The supine standard knee examination is supplemented with assessment of medial/lateral and proximal/distal patellar displacement/glide, patellar tilt, apprehension with displacement, medial reduction testing, and documentation of specific sites of pain. In addition, muscle bulk, strength (including core), and flexibility are assessed along with pelvic tilt.

Iliotibial band tightness and hip abductor strength are assessed in the lateral position.

Finally, the prone examination evaluates quadriceps tightness, hip/femoral rotation, and foot progression. Foot progression angle is measured with the knee flexed to 90 degrees. A line through the foot is compared with the femur for external or internal foot progression angle.

IMAGING

Initial imaging for patients with suspected PF chondral disease is standard standing anteroposterior, 45 degrees posteroanterior, true lateral, and low flexion angle axial views (eg, Merchant views). The Merchant view will show the congruence of the patella and femoral trochlea, patellar tilt, subluxation, and may show evidence of joint space narrowing (only at this specific flexion angle) or osteophyte formation. The lateral view is used to evaluate patella alta or infera. If it is a true lateral, it is valuable in assessing trochlear dysplasia and patellar tilt.[8]

Computed tomography was historically important in assessing patellar and trochlear anatomy, but is supplanted by the MRI in light of radiation concerns. It remains valuable for range of motion and quadriceps active and nonactive studies, or when hardware or contraindications preclude an MRI. To assess the articular surfaces the study is enhanced with dye creating an MR arthrogram. MRI is more commonly used to assess cartilage, bone, and anatomic variants. The anatomic measurements include the Caton-Deschamps patellar height ratio, Insall-Salvati patellar height ratio, or Blackburn-Peele index, the patellar trochlear overlap index, tibial-to-femoral rotation, the popular tibial tuberosity-trochlear groove (TT-TG) distance and less well known tibial tuberosity-posterior cruciate ligament (TT-PCL) distance.[9] Parikh and Noyes[10] devised a protocol that allows hip-to-knee-to-ankle measurement to further assess femoral anteversion and tibial torsion contributions. It is important to be familiar with the ongoing evolution of both the measurement of these studies,[11] as well as use of these studies (certainly the historical cut off of 20 mm TT-TG measured by computed tomography[12] must be tempered with entire hip to ankle considerations).

Furthermore, MRI allows sizing of the chondral defect as well as measurement of the depth and any associated subchondral bone marrow lesion.

NONSURGICAL TREATMENT

Nonsurgical management is used as an initial treatment modality to treat chondral lesions of the PF joint for at least 6 months or longer to reverse defects of strength, balance, and flexibility.[13] Treatment includes nonsteroidal anti-inflammatory medications, intra-articular corticosteroid injections, viscosupplementation, physical therapy, taping, and bracing. Physical therapy should first aim at restoring range of motion and flexibility followed by strengthening exercises to include core, pelvis, and quadriceps. In a study by Chiu et al,[14] PF pain improved with isometric and isokinetic weight training focusing on quadriceps strengthening, yet most therapists now emphasize a comprehensive approach that is "Core to Floor."[15]

SURGICAL MANAGEMENT

When patients with PF chondral injuries have failed conservative measures, surgical management may be considered. Some patients may appear to be candidates "on paper," yet are not in light of psychosocial, weight reasons, or unrealistic expectations. Successful surgical treatment is dependent on a thorough understanding of the underlying pathomechanics from "Core to Floor."[15] Before embarking on PF cartilage restoration, a thorough knowledge of all current PF surgical options is imperative. The key to optimal outcomes with PF cartilage restoration is optimizing the PF biomechanical environment and may often include balancing proximal, medial, and lateral soft tissues, tuberosity position, or even remotely such as femoral derotation or distal femoral varus osteotomy.

Cartilage-specific procedures may be divided into: palliative, reparative, restorative, and reconstructive methods. Palliative techniques, including loose body removal or debridement of the defect, attempt to relieve mechanical symptoms while obviously not restoring the articular surface. True reparative techniques fix chondral/osteochondral fragments. Restorative techniques, endogenous cell therapy (marrow stimulation and augments) and exogenous cell therapy [autologous chondrocyte implantation (ACI) or particulate juvenile cartilage], share the goal of using cells to restore the defect. Reconstructive methods use either autograft or allograft transplanted into the osteochondral lesion to fill in the bone and articular cartilage defect. The specifics of each of these treatments is beyond the scope of this article, however we will focus on pearls, pitfalls, and outcomes related to PF lesions.

MICROFRACTURE/MARROW STIMULATION

Microfracture, as first described by Steadman, is indicated for small ($< 2.5\,cm^2$) full-thickness cartilage defects and involves debridement of cartilage within the lesion down to subchondral bone. Awls are then used to create perforations in the subchondral bone plate. Perforation of the subchondral bone results in extravasation of blood and marrow elements with formation of a blood clot in the defect. Pluripotent, marrow-derived mesenchymal stem cells then migrate into the clot and promote the formation of a fibrocartilaginous repair tissue.[16–18] Technically, trochlear lesions may be accessed arthroscopically; however, patellar lesions may require a mini-arthrotomy to obtain the correct angle for the awl to penetrate the subchondral bone. Steadman et al[19] found significant improvement in pain and knee function after microfracture at an average of 11.4 years. Of the patients assessed in this follow-up study, 20 lesions were noted to be in the trochlea, and 8 were noted to be in the patella. However, the outcomes following microfracture tend to decline with time. A review of microfracture by Mithoefer et al[20] demonstrated that the procedure resulted in initial improvement during the first 24 postoperative months in all 28 studies included in the analysis. However, 7 studies in this review reported deterioration of functional outcomes in 47% to 80% of

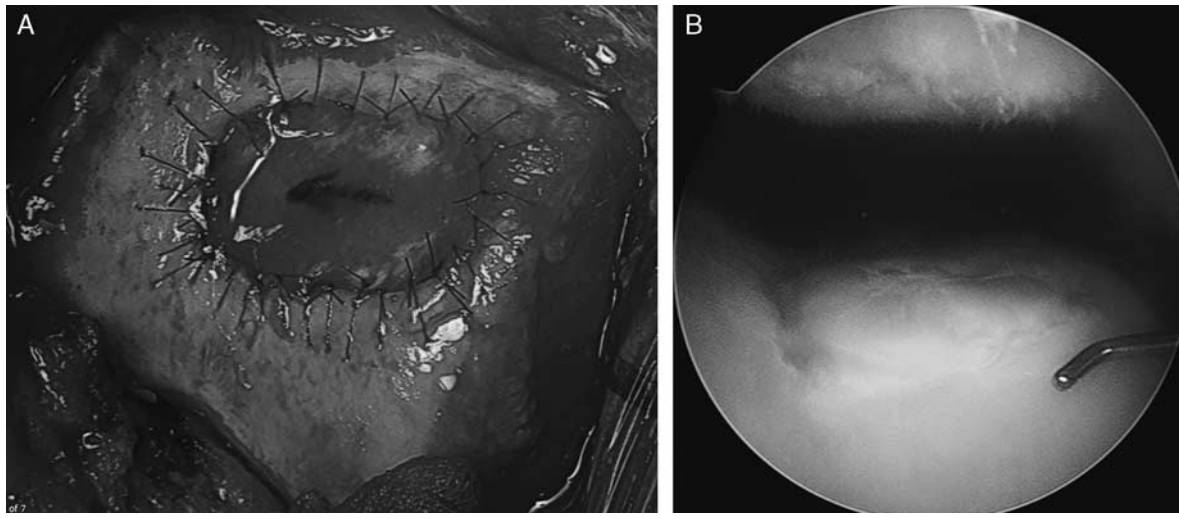

FIGURE 1. A, Central/lateral trochlear lesion measuring 20 mm medial-lateral and 18 mm proximal-distal treated with autologous chondrocyte implantation and tibial tuberosity straight anteriorization. B, Arthroscopy 9 months postimplantation showing full fill, full marginal integration, smooth surface with very mild overgrowth for an ICRS score of 12/12.

patients between 18 and 36 months after microfracture. Furthermore, Kreuz et al[21] reported poor results for all patellar lesions treated with microfracture. Marrow stimulation augments may offer advantages. Although long-term studies are needed, Gobbi et al[22] demonstrated encouraging results with BMAC augmentation.

Pearls

1. Thorough debridement of all damaged cartilage
2. Creation of stable vertical walls
3. Removal of the calcified cartilage layer

Pitfalls

1. Disruption of the subchondral plate if <2 to 3 mm between holes

ACI

ACI involves a diagnostic arthroscopy, harvesting a small amount of cartilage for cell culture, and a second stage in which the cells are reimplanted into the defect. It is most effective for medium and large size lesions. The cells are obtained from a non–weight-bearing region of the knee and then grown in vitro for 4 to 6 weeks. At time of reimplantation, the calcified cartilage layer is debrided, and the defect is covered with a synthetic membrane that is sutured in place. Fibrin glue is used to seal the edges and the cells are injected beneath the patch.

Early results of ACI for patellar lesions found poor results in patients that did not undergo any treatment for an underlying biomechanical knee disorder.[23] However, when ACI was performed in conjunction with a tibial tubercle osteotomy the results improved, with significant improvements in functional outcomes and patient satisfaction[24] (Fig. 1). This has been replicated by a number of studies: ACI in combination with anteromedialization (AMZ) has been shown to have superior outcomes, as compared with ACI alone, with 86% of patients having

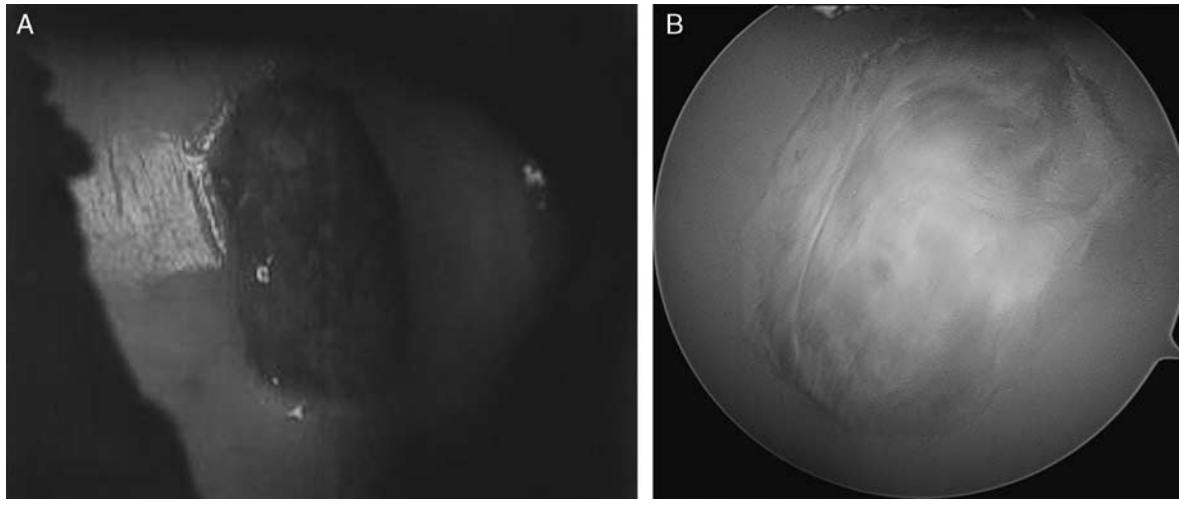

FIGURE 2. A, Central trochlear lesion measuring 12 mm medial-lateral and 20 mm proximal-distal treated with particulated juvenile allograft cartilage (DeNovo NT). B, Arthroscopy 2 years postimplantation showing full fill, full marginal integration, largely smooth surface with light fibrillations at margins for an ICRS score of 11/12.

good to excellent results following the combined procedure versus 55% to 65% of the patients treated with ACI alone.[25–28] Recent studies[29,30] have highlighted the good short-term and mid-term outcomes of ACI in patients with PF defects. A multicenter study of 110 patients treated with ACI demonstrated good to excellent results in 86% of patients at an average of 7.5 years follow-up.[29] Sixty-nine percent of the patients underwent a concurrent AMZ procedure, and 27% of the patients had bipolar disease. Similar results were shown by Gillogly and Arnold,[30] with good to excellent results in 83% of patients, all of whom underwent AMZ at the time of ACI. These studies highlight the need to correct alignment in conjunction with any cartilage procedure to improve results.

Pearls
1. Oversize the membrane to match the contour of the patella or trochlea
2. Address alignment preoperatively

Pitfalls
1. Not excising enough diseased cartilage
2. Tying knots too loosely
3. Injecting cells before adequate hemostasis or watertight seal

PARTICULATED JUVENILE CARTILAGE ALLOGRAFT (DENOVO NT)

Particulated juvenile cartilage allograft (DeNovo NT) (Zimmer Biomet, Warsaw, IN) is a relatively new technique for filling chondral defects. Studies have demonstrated that chondrocytes from particulated cartilage can migrate to form new hyaline-like repair tissue that integrates with surrounding tissue.[31] Theoretical advantages of DeNovo NT over ACI is that it uses juvenile cartilage noting these chondrocytes are more metabolically active, thereby possibly producing more extracellular matrix; and the procedure can be performed in a single-stage although a staging scope is needed to grade and size the lesion to allow ordering the product.[32]

The technique for DeNovo NT includes a standard parapatellar arthrotomy, followed by preparation of the defect with removal of any remaining cartilage and leaving vertical walls at the periphery. Implantation of the cartilage pieces into the defect is followed by covering with fibrin glue. This procedure is done in conjunction with a tibial tubercle osteotomy if indicated (Fig. 2).

There are limited results of particulated juvenile cartilage allograft. One study of 9 patients treated with DeNovo NT showed significant improvement in functional outcome scores at the 12- and 18-month follow-up time points.[33] Another study of 9 patients with patellar lesions treated with DeNovo NT demonstrated overall KOOS score improvement from a mean of 58.4 to 69.2 ($P = 0.04$) at 8-month follow-up.[31] Six of these patients underwent concomitant AMZ of the tibial tubercle. A study of 15 full-thickness patellar lesions treated with particulated juvenile cartilage allograft found a mean fill of the defect of 89%, whereas 2 patients required debridement for graft hypertrophy.[34] The study concluded that particulated juvenile articular cartilage allograft offers a viable option for patients with focal grade 4 articular cartilage defects of the patella.

Pearls
1. Decision process follows classic autologous chondrocyte implantation
2. Lesion preparation same as autologous chondrocyte implantation
3. May have theoretical advantage, if patient suspected of having poor cartilage genetics

Pitfalls
1. Undergrowth, overgrowth, and delamination possible
2. Uncontained lesions will require a patch
3. Considered experimental by many insurance carriers and therefore do not pay

OSTEOCHONDRAL AUTOGRAFT TRANSFER

Osteochondral autograft can be performed with several commercially available systems that harvest OC plug(s) from a low–weight-bearing surface of the knee to replace the chondral defect. It has had success in the treatment of femoral condyle chondral lesions; however, such success in the PF joint has been variably reported. This is due to the difficulty matching the plug to the complex architecture of the patella and trochlea. The clinical outcome of osteochondral autograft has mixed results in the literature. Hangody and Füles[35] reported on 10-year clinical outcomes and found 79% good to excellent results for patellar lesions. Astur et al[36] recently published on 33 patients with patellar chondral lesions treated with osteochondral autograft and found significant improvement in functional scores 2 years after surgery.

Pearls
1. Perpendicular placement of the harvesting instrument
2. Ensure recipient site is appropriate depth

Pitfalls
1. Excessive force during plug seating may kill chondrocytes
2. Leaving plug proud

OSTEOCHONDRAL ALLOGRAFT TRANSPLANTATION

Osteochondral allograft transplantation is a salvage procedure that can be used to treat large ($> 4\,cm^2$) chondral lesions. A fresh matched cadaver graft is used to restore the PF joint. This can be performed as a single-staged procedure and is useful for patients who have failed treatment with another cartilage repair technique.[37] Results of patellar and PF allograft replacement have demonstrated graft survival in 39% to 78% of patients with follow-up to 10 years.[37–40] Meric et al[39] demonstrated a lower survivorship in bipolar lesions compared with focal lesions. Gracitelli et al[40] found good results in 28 knees treated with PF allograft for isolated patellar lesions with survivorship of 78% at 5 and 10 years.

Pearls
1. Careful inspection of entire joint to ensure tibiofemoral compartment has no disease
2. Restore original patellar thickness

Pitfalls
1. Lateralizing the patellar component
2. Not assessing patellar tilt or subluxation

PF ARTHROPLASTY

PF arthroplasty is an option for patients with isolated PF arthritis or as a salvage procedure for failed cartilage

repair when salvage with OC allograft is not optimal. Improved outcomes have been seen in patients with trochlear dysplasia, but no maltracking.[41] The ideal patient age range for PF arthroplasty has yet to be determined, but some investigators believe that it is best restricted to patients younger than age 60 years who have isolated PF arthrosis.[41] Early failures are most commonly related to patellar maltracking. This has improved with newer prosthesis implant designs. The common cause of long-term failures is progressive tibiofemoral arthritis.[42–45] A recent study of mid-term outcomes by Goh et al[46] found 76% good outcomes, and a 92% survival rate in 51 patients treated with PF arthroplasty.

Pearls
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CONCLUSIONS

PF pain is a common musculoskeletal complaint. The majority of these patients can be successfully managed with physical therapy. A small number of patients with symptomatic chondral defects that fail nonoperatively will undergo surgery.

Treatment of underlying malalignment, instability, trochlear dysplasia is essential to a successful outcome. The cartilage defect may be managed with microfracture, ACI, particulated juvenile cartilage allograft (DeNovo NT), osteochondral autograft, osteochondral allograft, or PF arthroplasty depending on the size of the defect and patient characteristics. This topic remains a difficult and controversial disease to treat.

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