Management of Patellar Chondral Defects

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Abstract

Patellar cartilage has a poor capacity for healing because of the avascular and hypocellular nature of articular cartilage. Surgical options for cartilage defects are varied and include repair, regeneration, and reconstruction. Open reduction internal fixation of chondral defects should be attempted when a large chondral fragment with bone is present. This is frequently seen following patellar dislocation, patellar fracture, or in the setting of osteochondritis dissecans lesions. Cartilage regeneration options include microfracture and a bone marrow-stimulating technique that involves penetration of the subchondral bone. This technique is best for small, isolated defects. Augmentation to microfracture with biologically active adjuncts is becoming more widely available and is thought to enhance stem cell production and tissue regeneration. Cartilage reconstruction options such as autologous chondrocyte implantation area cell-based therapy that develops hyaline-like cartilage, as opposed to the fibrocartilage of microfracture, and has the added advantage of ease in contouring to patellar anatomy. Short-term data suggest improvement of clinical outcomes for most patellar cartilage techniques; however, long-term studies are needed to assess the durability and clinical outcomes of these evolving procedures.

Keywords: Patellar, Chondral, Cartilage

Introduction

The surgical treatment of patellar chondral defects is an ongoing challenge in orthopedics. Lesions of the patellar cartilage are common and can predispose patients to debilitating pain, dysfunction, and degenerative changes of the knee [1, 2, 3, 4, 5, 6, 7]. A review of 25,124 knee arthroscopies reported a 60% prevalence of chondral lesions with the patella being the most common location [8]. Most of these lesions are Outerbridge [9] Grade III or IV (Table 1).

There are many known causes of patellar cartilaginous damage. Chondral injuries can occur from acute trauma. This can be from a direct blow to the knee, such as in contact sports, a fall from height, or a dashboard injury such as experienced during a motor vehicle accident. Traumatic patellar dislocations can also predispose the chondral surfaces to abnormal translation and precipitate secondary damage. Nomura et al. [10] arthroscopically evaluated 39 patients with a mean age of 18 years (range 12–38) after acute patellar dislocation. 37 of the 39 (95%) individuals had patellar chondral defects, though acute versus chronic defects were not differentiated. Finally, chondral injuries can occur from abnormal stress, such as seen with lateral compression or excessive lateral positioning of the patella in the trochlear groove (TG) [5]. Over time, these abnormal forces can lead to cartilaginous damage. A more rare cause is osteochondritis dissecans [5]. Effective treatment of chondral lesions is difficult because of the avascularity and hypocellularity of the articular cartilage, which limits the healing potential [11]. Unless there is an acute need for surgery, as in the case of a large osteochondral loose body or fracture, management begins with a trial of conservative treatment, which focuses on a physical therapy program with attention to core muscle groups, attention to faulty body movement patterns, and improvement of knee range of motion. In cases of lateral patellar overload and other chronic conditions, there should be an assessment of the patient's body mass index (BMI) and fitness level, with appropriate intervention if BMI is high and/or fitness level is low.

Surgery is typically indicated after ongoing symptoms with non-operative management in full or nearly full-thickness chondral lesions. The surgical interventions aim to augment the durability of repaired defects and minimize further chondral damage [12]. Important considerations in surgical decision-making include defect size, location, condition of the subchondral bone, and unipolar versus bipolar lesions, as well as if prior cartilage procedures were performed [13]. A comprehensive understanding of the various treatment options, as well as their strengths, weaknesses, and outcomes, will help guide surgeons with treatment. Furthermore, this knowledge will assist in optimizing patient outcomes, decreasing the need for recurrent surgeries, and minimizing complications.

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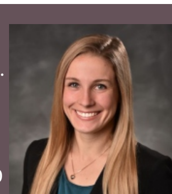

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Table 1: Outerbridge classification of chondral defects [9]

Grade	Criteria
0	Normal
I	Softening and swelling of articular cartilage
II	Fragmentation and fissuring in an area < 12 mm in diameter
III	Fragmentation and fissuring in an area more than 12 mm in diameter
IV	Erosion of cartilage to subchondral bone

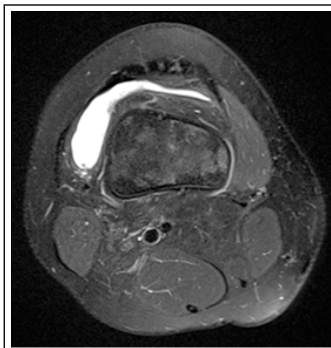

Figure 1: T2-weighted magnetic resonance imaging with effusion in the patellofemoral joint.

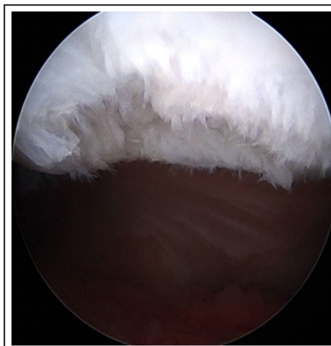

Figure 2: Arthroscopic image of osteochondral defect of the patella.

Clinical Vignettes

Traumatic

Osteochondral fracture without dislocation:

• A 14-year-old previously healthy female presented to clinic 2 months after a direct blow to the anterior aspect of her right knee during a soccer game. After the injury, she had symptoms of knee pain and swelling and an antalgic gait.

• On examination of her knee, 1+ effusion was present. Knee was stable to varus / valgus stress, with subtle patellar crepitation with flexion and extension. Lachman and pivot-shift maneuvers were negative. Patellar translation revealed stable patellofemoral ligaments. Magnetic resonance imaging (MRI) revealed a large osteochondral fracture on the chondral surface of the medial aspect of the lateral patellar facet as it abutted the central ridge, as well as 3 loose bodies ranging between 0.8 and 1.7 cm. A small effusion was present (Fig. 1). Further imaging revealed no anatomic patellar instability factors (APIF).

Direct trauma can cause all layers of the cartilage and subchondral bone to be injured. In less severe injuries, weakened cartilage may undergo repeated microtrauma that results in larger defects over time. Surgical interventions for osteochondral fractures include debridement, removal of loose bodies, fixation of large fragments, and marrow stimulating techniques such as microfracture and cartilage-restoration procedures such as autologous chondrocyte implantation (ACI), mosaicplasty, or osteochondral allograft transplant [14]. Demographic factors such as patient age, activity level, comorbidities, and local factors such as limb alignment, and stability of ligaments and menisci, should all be taken into consideration when deciding on the most appropriate management. This patient underwent loose body removal, chondroplasty of the patellar surface, and an ACI biopsy was obtained (Fig. 2 and 3). Depending on future symptoms, the ACI biopsy may be implanted into the chondral defect if symptoms persist.

Acute patellar dislocation with osteochondral injury

• A 14-year-old previously healthy female presented to clinic 2 months after tripping during basketball practice and falling on her left knee. She had immediate pain in the knee with moderate swelling. She denied any catching, locking, or popping. Her symptoms worsened with stairs and prolonged sitting. She had no history of prior patellofemoral injuries.

• On examination, she had a significant knee effusion. She was tender over the medial femoral condyle; there was no pain with patellar compression. Patellar translation was 1 quadrant medially and 2 quadrants laterally with a soft endpoint and (+) apprehension sign. Knee range of motion was 0°–130° with normal patellar tracking. Tibiofemoral examination was stable. She could perform a straight leg raise but was unable to actively extend the knee against gravity from a flexed position.

• Imaging showed a lateral patellar dislocation, evidenced by high-grade injury involving the femoral attachment of the medial patellofemoral ligament (MPFL) and typical bone bruises along the medial patella and lateral femoral condyle. There was a large joint effusion with a loose cartilaginous body from the apex of the patella measuring 0.6 cm × 1.7 cm × 2.3 cm. APIF (radiographs): A shallow TG (angle of 148° on low angle axial view), mild patella alta (Insall-Salvati ratio 1.3), and patella trochlear index (PTI) 0.25.

• Most patellofemoral surgeons agree that dealing with large (typically > 15 mm2) osteochondral patellar defects is necessary with removal versus fixation, depending on the quality of the lesion. Medial stabilization with MPFL reconstruction is more controversial due to the risk of arthrofibrosis. MPFL reconstruction is typically recommended for large chondral defects that have undergone repair (to protect the repair), when there is a recurrent dislocation, and/or when there are other patellar instability factors. The value of

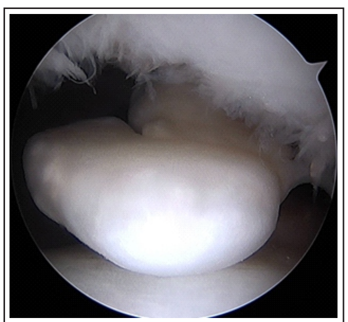

Figure 3: Loose body in patellofemoral joint seen with arthroscopy.

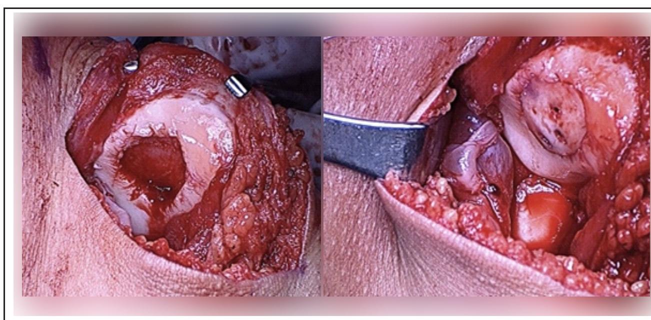

Figure 4: Intraoperative images with results of open repair of the central patella full-thickness chondral defect with headless compression screw fixation.

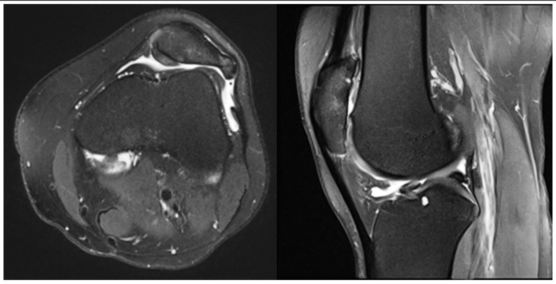

Figure 5: (a and b) Magnetic resonance imaging of axial and sagittal cuts demonstratinginferior lateral chondral wear to the patella and tight lateral retinacular structures.

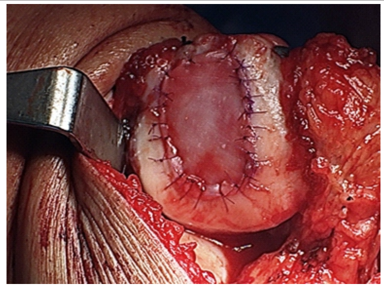

Figure 6: Intraoperative image of autologous chondrocyte implantation in the lateral patellar facet.

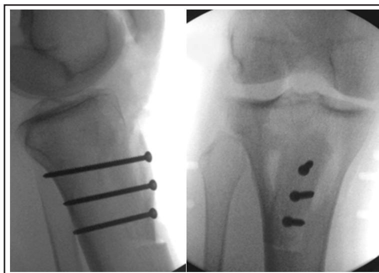

Figure 7: Lateral and AP post-operative x-ray images of tibial tubercle osteotomy with anterior medialization of the tubercle.

MPFL with an acute injury remains debatable. In this case, the patient presented with a subacute injury (2 months old at presentation) with a primary femoral based lesion with some signal change in the midsection; both favored against MPFL repair with cartilage stabilization.

• This patient underwent arthroscopic loose body removal; on inspection, it was felt to be reparable. Further, open repair of the central patella full-thickness chondral defect with headless compression screw fixation (Fig. 4), and MPFL reconstruction with hamstring allograft, was performed to provide improved patellar stabilization and decrease the risk of recurrent instability. Given the patella alta of 1.3 with satisfactory PTI, a distalization of the tibial tubercle (TT) was not performed.

Atraumatic

Patellofemoral overload / increased lateral patellofemoral stress:

• A 22-year-old previously healthy female presented to the clinic with a 3-year history of persistent right knee pain and swelling. Her symptoms worsened with participation in collegiate softball and persisted through a variety of conservative treatment options including activity modification, rest, anti-inflammatories, physical therapy, and a viscosupplementation injection. She had no history of previous patellofemoral dislocations or instability.

• On knee examination, a trace effusion was present with full knee range of motion. The patella had 2-quadrants lateral and 1-quadrant medial translation, both with firm endpoints and no apprehension. Patella tilt was to 10° short of neutral. Subtle crepitation and tenderness over her lateral patellar facet were present. MRI imaging (Fig. 5) revealed patellofemoral joint chondrosis with the full-thickness chondral loss, and moderate to large joint effusion.

As opposed to acute injuries, chronic lateral patellar overload presents as a more insidious cause of patellar chondral defects. Anatomic features such as genu valgus, trochlear dysplasia, patella alta, and/or increased factors including quadriceps angle, femoral anteversion, external tibial torsion, and TT-TG distance can predispose patients to increased lateral patellofemoral stress [15, 16]. Patients with cartilaginous injuries from abnormal patellofemoral stress frequently require operations that improve alignment and patellofemoral engagement in addition to operations that address chondral defects. In this patient, we proceeded with a 2-stage procedure. The first stage consisted of patellar chondroplasty and an ACI biopsy. When the patient's symptoms did not improve from the initial debridement and appropriate rehabilitation, the second stage procedure was performed. This consisted of ACI implantation in the lateral patellar facet (Fig. 6), lateral retinacular lengthening, and an anteromedial TT osteotomy (Fig. 7). A lateral lengthening was performed in this case as the patient's tight lateral reticular structures limited tilt of the patella to less than neutral. A TT osteotomy was performed with a relatively steep osteotomy to unload the inferolateral patellar facet.

Repair Techniques

Open reduction internal fixation

Fixation of loose osteochondral fragments, first described by Matthewson and Dandy [17] is an appropriate option for osteochondral fractures with large fragments, sufficient subchondral bone, and in weight-bearing regions [18]. Viable fragments should be repaired if able, especially in young patients [13]. Fixation techniques of osteochondral fractures can include the use of suture, metal screws, and bioabsorbable pins [14] (Arthrex Inc. Naples, FL). Partially threaded screws provide the best fixation; however, if they are not countersunk, they can irritate the articular surface and require later removal [18]. Headless screws obtain good fixation but can back out over time, thus may also requiring later removal [18]. Bioabsorbable implants are good for small lesions with minimal subchondral bone and do not require later removal; however, they are more expensive and provide less compression compared to the other devices [18]. While it has been thought that sufficient bone is essential for good outcomes with this procedure, it has recently been recognized that fixation of primarily cartilaginous fragments can be successful [13]. This procedure provides the benefit of restoring articulation of the patellofemoral joint to decrease the chance of long-term arthrosis.

Good clinical outcomes and radiographic congruency with a fixation for patellar osteochondral fractures have been reported at follow-up up to 5 years in patients from 11 to 74 years [15, 19]. Small case series have shown that open reduction internal fixation had better outcomes in other parts of the knee versus the patella [20, 21].

Regeneration Techniques

Chondroplasty

Arthroscopic debridement, or chondroplasty, is a straightforward intervention that can be used alone or in conjunction with another technique. Chondroplasty is particularly helpful in mild to moderate patellar chondral defects that are not yet full-thickness. Loose chondral tissue that may impinge normal articulation and calcified cartilage is removed [11]. Debridement can be used on large flap components and lesions that are staged to undergo a more extensive chondral repair procedure. This is a quick technique with a short post-operative recovery, however the effects of chondroplasty often diminish with time, thus longer follow-up is important to gauge the durability of this procedure [11].

Microfracture

The microfracture technique, first described by Steadman et al. [22], was proposed as a quick, easy, and cost-effective way to restore full-thickness, 1–3 cm2 chondral defects [22, 23]. This type of bone marrow stimulation technique creates multiple holes in the subchondral bone plate. All the damaged cartilage is removed to a rim of healthy cartilage, and the layer of calcified cartilage at the base of the defect is also removed to aid in clot formation [22]. The appropriate depth is confirmed when blood and fat droplets are visualized from the bone marrow cavity once inflow is let down. These contents possess mesenchymal cells that differentiate into fibrochondrocytes that are stabilized by clot formation. The biggest drawback of marrow-stimulating techniques is that the newly formed fibrocartilage, a form of Type I cartilage, has less mechanical stress resistance than native cartilage; thus, benefits of the technique may be short-lived [18, 22]. Furthermore, the number of stem cells procured is low and continues to decrease with age [1, 11]. Improvement in clinical outcomes has been reported in patients who underwent microfracture for patellar chondral defects, particularly in younger patients (< 30–40 years of age), defects < 4 cm2, when microfracture was used as a first-line procedure, BMI < 30, and higher preoperative activity levels [22, 24]. Despite these favorable outcomes, deterioration has been shown between 18 and 36 months after microfracture, with patellar defects faring significantly worse than femoral condylar lesions [25]. Worse outcomes in the patella are likely due to the bone quality of the patella and its status as a sesamoid bone. Furthermore, microfracture of the patella frequently necessitates an open approach, with the concern of increasing surgical morbidity and time to recovery. The posterior directed orientation of the patellar articular cartilage may also play a role, as clot formation may be inhibited as the patient lies supine following surgery.

Adjuncts to microfracture

Since the popularization of microfracturing, new scaffolding techniques have been developed to augment this procedure. These techniques utilize materials designed to enhance progenitor cell development. The microfracture combined with autologous matrix-induced chondrogenesis (AMIC) technique, first described by Behrens [26, 27], utilizes a porcine collagen Type III/I membrane to manage larger defects up to 9 cm2 [2, 5, 23]. The collagen matrix is a natural scaffold for cell attachment and acts as a catalyst for differentiation. Gille et al. [2] used AMIC for large (mean 4 cm2, range 1.3–8.8 cm2) Outerbridge Grade IV chondral defects and found significant clinical improvement at an average follow-up of 37 months (range 24–62 months).

Bone marrow aspirate concentrate (BMAC) provides stem cells and growth factors that are thought to enhance chondral repair in damaged areas [28, 29, 30]. Bone marrow from the iliac crest has been found to contain greater mesenchymal cells than either tibial or femoral bone marrow [29]. 60 mL of BMAC are harvested, placed on the chondral defect, and stabilized with either a collagen I/III or polyglycolic acid / hyaluronan scaffold [28, 29, 30]. This technique can be used to supplement microfracturing but has also been described as an isolated procedure [28, 30]. Studies have shown significant clinical improvement at 20–24 months follow-up [28, 30]. While most patients in these studies had 80% complete filling of chondral defect on MRI at least 10 months after surgery, many patients continued to have subchondral irregularities and non-homogeneous cartilage signal [28, 30].

BioCartilage (Arthrex Inc., Naples, FL) is a new product that has dehydrated, micronized allogenic cartilage and is inserted with plasma-rich protein (PRP) over chondral lesions that have been microfractured [31]. The anabolic and anti-inflammatory aspects of PRP are thought to help with tissue regeneration [32]. Preclinical studies have been underway that have shown promising results with BioCartilage for chondral defects; however, no human outcome data are yet available [31].

Cartilage Reconstruction Techniques

ACI

ACI, first described by Brittberg et al. [33], involves harvesting articular cartilage, culturing these chondrocytes to undergo proliferation, and transplanting chondrocytes into chondral defects. ACI is a common procedure for patellar chondral defects because of its ease in contouring to patellar anatomy. Due to this property, it is a preferred intervention for bipolar patellofemoral lesions, though results tend to be better with unipolar lesions [13]. Unlike microfracturing, which repairs defects with a fibrocartilaginous substance, ACI is a restorative technique that aims to replace chondrogenic cells and type II collagen present in native articular cartilage [11]. In addition, ACI preserves that subchondral bone plate which is disrupted with microfracture. ACI is a two-stage procedure. In the first stage, 200–300 mg of articular cartilage are biopsied arthroscopically from the osteochondral ridge of the superior medial or lateral femoral condyle, as these are minimal weight-bearing surfaces [34]. The biopsy contains hundreds of thousands of cells that undergo digestion, differentiation, and expansion to millions of cells. During the second stage, the defect is exposed,and the cells are re-implanted. First-generation ACI uses a periosteal patch sutured over the defect. Because of reports of high reoperation rates, second- and third-generation ACI techniques have been developed, which utilize a collagen membrane (C-ACI) and membrane-associated techniques [34, 35, 36]. Limitations include the two-stage nature of the procedure and its technical difficulty. In addition, reported outcomes have included prolonged postoperative effusion, hypertrophy of the periosteal patch, donor site morbidity, and failures in as many as 50% of cases [1, 25, 34, 35, 37, 38]. Larger defects and a history of previous microfracture increase risk for failure [38]. Patients with patellar chondral defects have been found to have favorable outcomes at an average follow-up of 38 months [39]. The generation of ACI technique did not affect results, and defects on the lateral facet of the patella had better clinical outcomes compared to other areas. Steinwachs and Kreuz [40] found no significant difference in outcomes after ACI with a Type I/III collagen membrane at 36-month follow-up, based on the location of defect in the knee (condyles, trochlea, and patella).

Osteochondral autograft transfer system (OATS) and mosaicplasty

Osteochondral autograft was first described by Outerbridge et al. [41] for the treatment of osteochondritis dissecans in the femur. OATS is a technique for treating full-thickness focal chondral defects between 1 and 4 cm2 when subchondral bone is compromised [3, 5]. Mosaicplasty, a term popularized by Hangody et al., [42, 43] involves transplanting multiple small and cylindrical osteochondral plugs over damaged cartilage. This intervention is thought to be better than ACI after failed patellar cartilage procedures. In this technique, cylindrical osteochondral grafts are obtained from minimal weight-bearing surfaces. Grafts are different sizes to enhance contouring of graft in the defect and to allow a 90–100% fill rate [3]. This is a difficult procedure for patellar chondral defects, as there is a lot of patellar shape variability; thus, contour matching is difficult. Other disadvantages include the comorbidities associated with a donor site, the limited availability of donor graft, the prolonged period of limited weight-bearing postoperatively, and the differences in orientation and mechanical properties between the donor and recipient cartilage [1, 44]. In addition, potential empty space located between plugs at the recipient site may hinder the quality of the repair [1]. Over a 1–10-year period, Hangody and Füles [3] found good to excellent results in 79% of 119 patients who underwent patellar and/or trochlear joint mosaicplasties for Outerbridge Grade III or IV chondral lesions. These results were inferior to outcomes in patients with femoral condyle implantations and tibial resurfacings, with good to excellent results in 92% and 87% of patients, respectively. Other studies have also reported worse outcomes in patellofemoral cartilage lesions compared to condylar lesions [45, 46].

Additional Procedures

TT osteotomy

Some chondral defects of the patella are a result of chronic patellar instability secondary to malalignment of the quadriceps mechanism. In these cases, addressing cartilage injury of the patella must incorporate procedures that realign the patellofemoral mechanism to minimize further damage. Lateral patellofemoral overload associated with a tight lateral retinaculum may be treated with release or lengthening of the lateral retinaculum. Patients with more severe malalignment, defined by an elevated TT-TG distance > 15 mm on computed tomography imaging, may benefit from a TT osteotomy [47]. An anteromedial osteotomy, described by Fulkerson in 1983 [48] as a treatment for "persistent patellofemoral pain associated with patellar articular degeneration," decreases the Q-angle to a more centralized position through the anterior and medial transfer of the TT. Patients with proximal and medial facet patellar lesions have been shown to have significantly worse outcomes than those with distal and lateral facet lesions [49]. Diffuse patellar lesions and associated central trochlear lesions are also correlated with worse outcomes [49].

Conclusions

Managing chondral defects of the patella are difficult for orthopedic surgeons because of the limited healing potential of cartilage and the frequency of early cartilage breakdown in this region. This, combined with associated dysplastic variants common to this joint, creates challenges in formulating surgical guidelines. Many studies have reported fair to good outcomes following patellar cartilage repair [2, 3, 4, 12, 15, 22, 28, 30, 36, 39]; however, studies have also shown that the clinical outcomes deteriorate with time [4, 14, 24, 38]. It is difficult to compare various techniques because of the lack of well-designed randomized controlled trials in the current literature [1]. Patients who tend to have the best outcomes are younger, have lower BMIs, have unipolar defects < 4 cm2, and have not had previous cartilage repair surgeries [24, 30, 34, 38]. Furthermore, patients with lesions of the lateral and distal patellar facets are more likely to have better outcomes than patients with medial, proximal, or diffuse lesions [49] due to surgical techniques that can unload this region.

There is no general consensus of the best treatment for patellar chondral defects. However, the most important aspect of managing patellar chondral defects is to treat each patient individually, taking into account lesion size, location, the presence of chondromalacia, as well as concurrent conditions, including patellar alignment and soft tissue injury. APIF are essential to document and consider in all patellofemoral cartilage injuries.

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