Chondral Injury in Patellofemoral Instability

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Abstract

Objective: Patellofemoral instability is common and affects a predominantly young age group. Chondral injury occurs in up to 95%, and includes osteochondral fractures and loose bodies acutely and secondary degenerative changes in recurrent cases. Biomechanical abnormalities, such as trochlear dysplasia, patella alta, and increased tibial tuberosity-trochlear groove distance, predispose to both recurrent dislocations and patellofemoral arthrosis. *Design:* In this article, we review the mechanisms of chondral injury in patellofemoral instability, diagnostic modalities, the distribution of lesions seen in acute and episodic dislocation, and treatments for articular cartilage lesions of the patellofemoral joint. *Results:* Little specific evidence exists for cartilage treatments in patellofemoral instability. In general, the results of reparative and restorative procedures in the patellofemoral joint are inferior to those observed in other compartments of the knee. *Conclusion:* Given the increased severity of chondral lesions and progression to osteoarthritis seen with recurrent dislocations, careful consideration should be given to early stabilisation in patients with predisposing factors.

Keywords

patellofemoral joint, articular cartilage, instability

Introduction

The articular cartilage of the patellofemoral (PF) joint is the thickest in the human body, with patella cartilage thickness up to 7.5 mm and trochlea cartilage thickness up to 3.5 mm measured in normal subjects.[1] The joint is subjected to large compressive loads of up to 8 times body weight during squatting[2] and 5972 N when landing after a jump.[3]

The incidence of first time patellofemoral dislocation is 7 per 100,000 across all age groups,[4] but as high as 107 per 100,000 between ages 9 and 15 years.[5] Dislocation may occur in normal knees secondary to trauma; however, patients are frequently found to have predisposing factors such as trochlear dysplasia, patella alta, increased tibial tuberosity-trochlear grove (TT-TG) distance or ligament hyperlaxity.[6,7] Acute dislocation almost uniformly damages the soft tissue restraints,[8] in particular the medial patellofemoral ligament (MPFL), which provides 50% to 60% of resistance to lateral displacement.[9,10] Further dislocations, termed episodic patella dislocations (EPDs), occur in 40% to 70% with nonoperative management.[11-13] Chondral damage has been reported in more than 90% of patients with EPD.[14]

Pathogenesis of Chondral Injury

Chondral damage in patellofemoral dislocation may result from acute traumatic events, altered joint loading caused by ongoing instability and preexisting anatomical abnormalities, and iatrogenic injury due to surgical intervention.

Acute, macroscopic chondral injury includes osteochondral or chondral fractures and fissures occurring at the time of dislocation or reduction. These injuries may be detected in up to 95% of acute dislocations.[15] Resultant loose bodies may be visible on radiological investigations, and have traditionally been regarded as an indication for early operative intervention.[7] Loose bodies have been reported in 31% to 58% after a first dislocation.[16-18] The high incidence of osteochondral as opposed to chondral fractures in this generally young group may be due to age related properties of the subchondral bone, calcified cartilage, and cartilage layers.[19]

Ongoing chondral damage occurs after the acute injury, and involves cell death and extracellular matrix degradation. Cell death occurs by both chondrocyte necrosis and apoptosis,[20] and continues for 48 to 96 hours after injury.[21,22] Chondrocytes may undergo a specific type of apoptosis, termed chondroptosis, and the intracellular signaling pathways may differ from those seen in other cell types.[23]

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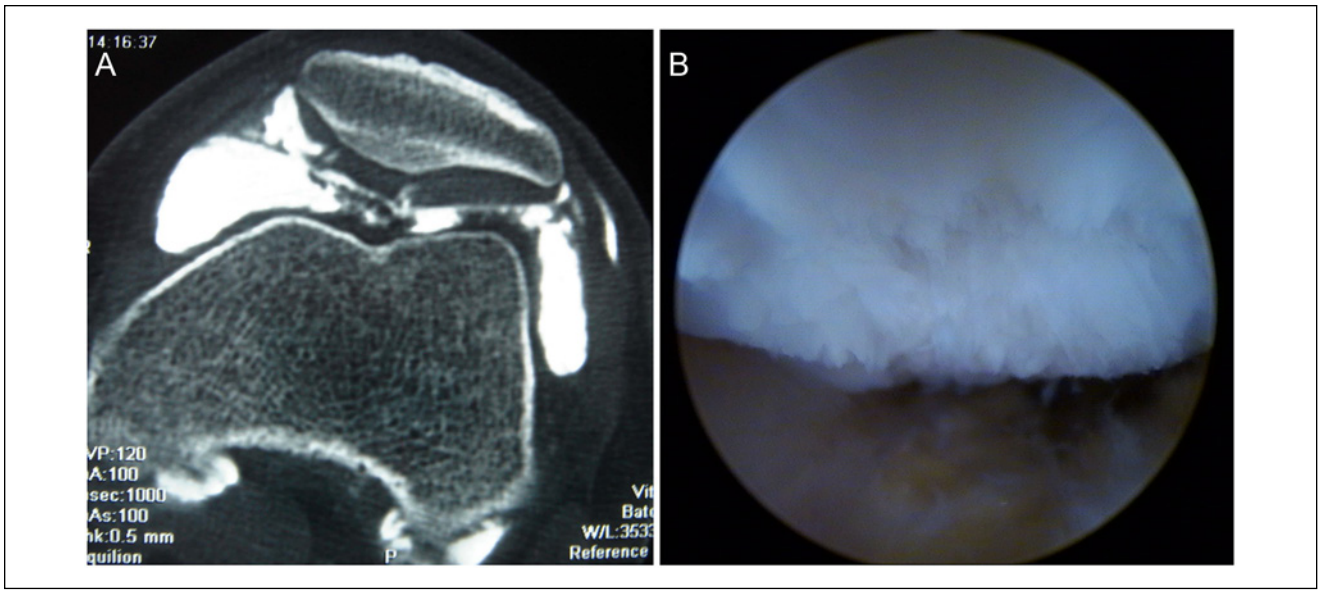

Figure 1. (**A**) Computed tomography arthrogram demonstrating central and medial patella chondral damage with loose cartilage in the patellofemoral joint. (**B**) Arthroscopic view in the same patient demonstrating chondral injury to the central and medial patella with loss of the patella ridge.

Extracellular matrix degradation occurs because of upregulation of matrix metalloproteinases. Numerous chemical mediators are involved in this process, including mitogen-activated protein kinases, p38, extracellular signal-regulated kinases, and c-Jun N-terminal kinases.[24]

Delayed chondral damage, or posttraumatic osteoarthritis (OA), occurs in the setting of chronic loading abnormalities.[25] Despite the long-standing clinical association between joint injury and subsequent arthrosis, the exact processes involved are poorly understood. Joint instability has been shown to increase peak contact stresses *in vitro*,[26] and the degree of instability to correlate with cartilage degeneration in an animal model.[27] Stephen *et al*.[28] have recently demonstrated a significant increase in lateral facet peak contact pressure in early flexion after sectioning of the MPFL, associated with increased lateral patella tilt and translation. Lateral tilt and translation are predictive of PF OA progression.[29] Preexisting joint structural abnormalities are also associated with cartilage damage. In hip dysplasia, cumulative contact stress–time exposure correlates with the development of secondary OA, with more than 90% risk for exposures over 10 MPa-years compared with only 20% below this level.[30] Jungmann *et al*.[31] have demonstrated trochlear dysplasia to be associated with more severe patellofemoral OA in 304 patients from the Osteoarthritis Initiative longitudinal study. Indeed, Noehren *et al*.[32] found the radiological risk factors for lateral PF degenerative disease in young patients to be almost identical those for PF instability, including trochlear dysplasia, patella alta, and increased TT-TG distance.

Surgical treatment of PF instability has been associated with secondary OA, increased pain, and even medial patella dislocation.[33] Direct chondral injury may occur during surgery, for instance, because of drill penetration. Patella fracture has been reported after MPFL reconstruction.[34] Medial PF degeneration has been demonstrated in long term follow-up after patella realignment surgery[35,36] and may be due to excessive medialization.[37] Increased medial PF joint pressures have also been documented with small increases in tension in MPFL reconstruction[38]; however, these techniques may result in less secondary degenerative change than realignment procedures.[39,40]

Diagnosis of Chondral Lesions

Chondral lesions in acute and episodic patella dislocation may be diagnosed radiologically or at operation by either arthroscopy or arthrotomy.

Plain radiographs provide valuable information regarding predisposing factors in PF instability and the development of arthrosis over the longer term; however, they are poor for the detection of osteochondral injuries. Stanitski and Paletta[18] found only 32% of arthroscopically proven lesions diagnosed on preoperative radiographs. Similarly, Dainer *et al*.[41] discovered osteochondral defects not visible on plain radiographs in 40%.

Similar to plain radiographs, computed tomography (CT) gives poor cartilage detail but allows assessment of other indices such as TT-TG. CT arthrography (**Fig. 1**), however, allows diagnosis of chondral injury with similar accuracy to magnetic resonance imaging (MRI) and may be more accurate in previously operated knees.[42]

Magnetic resonance imaging is able to diagnose chondral and osteochondral lesions noninvasively. Medial soft

tissue injuries and bone bruising patterns characteristic of patella dislocation may also be apparent,[43] as well as unsuspected ligament and meniscal injuries.[16,44] A recent systematic review found MRI to be 87% sensitive and 86% specific in diagnosing patella chondral lesions, and 72% sensitive and 89% specific for trochlear lesions when compared with arthroscopy.[45] von Engelhardt *et al*.[46] compared the accuracy of MRI diagnosis with that of arthroscopy in acute and episodic dislocations. They found MRI to be more accurate in acute compared to episodic cases. Accuracy was also better for higher grade lesions. Other authors, however, have reported false positives,[47] and found MRI to underestimate the size of cartilage lesions after debridement.[48]

Specific cartilage sequences, such as dGEMRIC (delayed gadolinium-enhanced MRI of cartilage) and T2 mapping, may have a role in the diagnosis and management of patellofemoral cartilage injuries in the future,[49-51] as may newer technologies, such as delayed quantitative CT arthrography (dQCTA)[52] and SPECT/CT (single photon emission computed tomography with conventional computed tomography).[53,54]

Arthroscopy remains the gold standard for diagnosis of chondral lesions of the PF joint.[45,47,55] When performed in conjunction with stabilization procedures, it allows diagnosis of meniscal and other internal derangements which may be missed at arthrotomy.[56] Arthroscopy is, however, an invasive surgical procedure, and the role of operative intervention in first time dislocation remains controversial.[13,57-59]

Distribution of Chondral Lesions

Damage to the patella articular cartilage has been reported in up to 95% of first time dislocations[15] and 96% in EPD[14]; however, most authors report rates between 70% and 80%.[16,18,56,60] The central and medial areas are the most commonly affected. The injury may occur because of shear at the time of dislocation as the patella moves over the lateral aspect of the femur, by impaction against the femur in the dislocated position, or by a combination thereof during reduction. Luhmann *et al*.[56] examined 41 knees arthroscopically, 7 after acute dislocations and 34 with EPD. They found patella lesions in 30 (73%), of which 16 involved the apex, 12 the medial facet, and 2 the inferior pole. The average lesion size was 112 mm^2 (25-256 mm^2). Vollnberg *et al*.[60] studied MRI findings in 129 knees divided into 3 groups: acute dislocations, recurrent dislocations (2-9 episodes), and chronic dislocations (>10 episodes). They found 79.8% had patella chondral lesions. The central region was affected in 75% overall, with 51.9% of these involving both the central and medial areas. Interestingly, they found the medial facet involved most frequently in acute events, but an increasing rate of lateral facet involvement with recurrent dislocations, suggesting the development of PF OA. They also found more chondral damage and other signs of OA with increasing numbers of dislocations. Nomura *et al*.,[14,15] in arthroscopic studies in both acute and episodic dislocations, found that patella chondral lesions were almost universal and always occurred in the inferior half to two-thirds of the patella. These inferior lesions may relate to patella alta, with the patella only partially engaged with the trochlea at the time of dislocation. They also found predominantly medial lesions in acute cases and more lateral involvement in episodic dislocation; however, there was no development of new lateral lesions in a subsequent second-look study in either group.[61] There was more progression in the EPD group.

Lesions affecting the cartilage on the femoral side are less common, with reported rates from 5% to 32%.[15,16,56,60] Interestingly, this is opposite to the pattern of bone bruising found on MRI, where the lateral femoral condyle is affected much more frequently than the patella.[43] Femoral lesions almost always occur in combination with patella lesions, although they have been reported to occur in isolation in up to 5%.[60] The majority of femoral lesions affect the lateral aspect of the lateral femoral condyle or trochlea, however, the weightbearing surface of the lateral condyle may also be affected.[62] Mashoof *et al*.[63] described 7 cases of osteochondral injury to the weightbearing portion of the lateral femoral condyle, ranging in size from 1.5 to 6.75 cm^2.

Medial PF joint degeneration has been reported following both proximal and distal realignment procedures.[35,36] The acutely damaged medial patella cartilage may be especially sensitive to the significant force increases seen with excessive medialization, particularly in the absence of medial femoral condylar hypoplasia.

Of note, associated injuries are commonly reported in both MRI and arthoscopic studies with an incidence of 11% to 31%.[16,44,56] Meniscal injuries, collateral ligament injuries and even partial anterior cruciate ligament tears have been reported.

Treatment Options

Treatment options depend on the type, location, and chronicity of the chondral lesion (**Table 1**) .

Nonoperative Therapies

Nonoperative treatment, including analgesic medication, physiotherapy and intra-articular injections of corticosteroids, is the first line of treatment. Viscosupplementation has been shown to reduce pain and improve function in PF OA.[64] The exact mechanism of action is unclear. *In vitro* research has suggested a disease modifying effect[65,66]; however, a recent trial using dGEMRIC MRI in the tibiofemoral compartments failed to demonstrate any structural or compositional change in the articular cartilage.[67] The role of viscosupplementation in acute cartilage lesions and instability is unknown.

Table 1. Chondral Lesions and Treatment Options in Patellofemoral Instability.

Instability Type	Lesion Location	Lesion Types	Cartilage Treatment Options
Acute	Medial/central > lateral patella	Osteochondral/ chondral fractures	Internal fixation
	Lateral femoral condyle	Loose bodies	Debridement
	Lateral trochlea	Chondral cracks	Microfracture
		Chondral flaps	Mosaicplasty
			Autologous chondrocyte implantation ± treatment of instability/osteotomy
Episodic	↑Lateral patella	As above, plus	As above, plus
	Lateral trochlea	Chondral fissuring	Bulk allograft
		Chondral fibrillation	Arthroplasty
		Chondral erosion	
		Osteophyte formation	

Cartilage Procedures

Osteochondral fractures may be amenable to reduction and internal fixation. A variety of fixation methods may be used, including conventional metal screws, headless compression screws, and bioabsorbable pins. Chondrocytes in osteochondral fragments undergo significant necrosis and apoptosis, which may affect the outcome of fixation procedures.[20] Lee *et al*.[68] found that patients undergoing internal fixation of patella osteochondral fragments did worse than those without fixation, although this may be explained by the larger size of the internally fixed lesions. Seeley *et al*.[17] found the functional outcome for fixation of osteochondral lesions following PF dislocation to be worse when the weightbearing portion of the lateral femoral condyle was affected[17].

Chondral defects may be treated by reparative techniques, such as marrow stimulation and osteochondral transplantation, or restorative procedures such as autologous chondrocyte implantation. In general, the results of these techniques in the PF joint have been inferior to other compartments of the knee.[69-71] Some data exist regarding cartilage procedures in association with PF realignment, although instability is rarely specifically identified as a indication or prognostic factor.[72-74] No study mentions ongoing instability after cartilage surgery. It makes inherent sense that repair techniques would have poorer outcomes in the setting of ongoing instability.

Marrow stimulation techniques, such as microfracture, Pridie drilling, and abrasion arthoplasty, rely on blood clot containing mesenchymal stem cells from bone marrow to fill chondral defects. These techniques are inexpensive, however, the resultant repair tissue is biomechanically inferior to hyaline cartilage, larger lesions do poorly and results tend to deteriorate over time.[75,76] While generally considered safe, complications such as intralesional osteophytes do occur.[77] In a study investigating the effect of patient age on microfracture results, Kreuz *et al*.[69] found PF lesions were most likely to deteriorate regardless of age. Periosteal flaps have been used to contain repair tissue, with improved results demonstrated over abrasion techniques for the patella.[78] More recently, scaffolds have been employed to improve the quality of repair tissue. Porcine collagen and synthetic polymer scaffolds coupled with growth factors and bone marrow concentrates are already in use.[79,80] In a multicenter randomized clinical trial using the scaffold-based BST-CarGel treatment compared with microfracture alone for femoral condylar lesions, Stanish *et al*.[81] demonstrated superior lesion filling and repair tissue quality at 1 year, although clinical superiority was not shown. Multiphasic and gradient scaffolds are also being developed aiming to recreate the tidemark and cartilage–bone interface.[82]

Osteochondral transplantation, such as mosaicplasty, can result in high-quality repair tissue with rapid healing; however, donor site morbidity limits the defect size that may be treated.[83] Hangody *et al*.,[84] reporting their 10-year experience with the mosaicplasty technique, reported 79% good-to-excellent results in 118 procedures involving the PF compartment, compared with 87% to 92% elsewhere in the knee. Gawęda *et al*.[72] studied the results of 19 patients with EPD and significant patella chondral defects (International Cartilage Repair Society [ICRS] grades III and IV) treated with single stage extensor realignment and autologous osteochondral grafting. Compared with a control group treated with realignment only (chondral lesions ICRS grades I and II), the treatment group had significantly worse preoperative Marshall scores (36.3 ± 2.1 vs. 40.7 ± 3.7) but comparable results at 24 months (46.2 ± 1.8 vs. 47.1 ± 1.6). Osteochondral bulk allograft may also be used in the PF joint, however, this should be viewed as a salvage procedure, with high rates of failure due to graft non-incorporation.[85,86]

Autologous chondrocyte implantation (ACI) is a 2-stage procedure involving chondrocyte harvest, expansion by

culture and subsequent reimplantation. These techniques offer the potential for more durable repair with true hyaline cartilage tissue.[87] The techniques have evolved from liquid chondrocyte solution injection under a sutured periosteal flap to the greatly simplified implantation of a biodegradable, chondrocyte seeded matrix, although only weak evidence supports the newer generations to date.[88] In 1994, Brittberg *et al*.[71] reported good-to-excellent results in only 2 of 7 patients treated for patella lesions with first-generation techniques. Bentley *et al*.,[70] in a randomized control trial of ACI versus mosaicplasty, reported 85% excellent or good clinical results for ACI compared with 60% for mosaicplasty for patella lesions using the modified Cincinatti rating system, although this did not reach statistical significance. Gobbi *et al*.[89] reported results for 38 patients treated with second-generation ACI (Hylograft C) for lesions of the PF joint, 17 of whom had previous or simultaneous patella realignment procedures. They reported 32 of 34 objectively normal or near normal knees at 2 years, and 31 of 34 at 5 years. Patients with patella lesions, however, had a significant decline in International Knee Documentation Committee (IKDC) scores from 2 to 5 years. Filardo *et al*.[90] recently reported comparative results for patella and trochlear lesions treated with matrix-assisted autologous chondrocyte transplantation. Patients with PF malalignment were excluded. They found better results in trochlear lesions than patella lesions at 5-year follow-up, with IKDC scores of 89.6 ± 12.7 and 69.7 ± 17.6, respectively.

A number of factors may influence the result of ACI procedures. Kreuz *et al*.[91] demonstrated inferior outcomes in female patients and for patella lesions, despite the exclusion of PF malalignment. Filardo *et al*.[92] found that females had not only lower raw IKDC scores following matrix-assisted autologous chondrocyte transplantation but also a higher incidence of patella lesions, and that the sex difference was not apparent after matched-pair analysis. Minas *et al*.[77] have demonstrated an increased failure of ACI procedures after previous marrow stimulation techniques, with failure rates three times that of untreated lesions. Pascual-Garrido *et al*.,[93] however, found no difference in outcome for those with previous cartilage surgery, although more than half the patients in their study underwent simultaneous anteromedialization (AMZ) of the tibial tuberosity. Platelet-rich plasma may also have a role in improving repair tissue quality.[94]

Of particular relevance to PF instability, Niemeyer *et al*.[95] demonstrated significantly better results for lateral facet lesions, compared with lesions involving the medial or both facets, as seen most commonly after acute PF dislocations. They have also described a "double eye" technique for ACI treatment of patella lesions to maintain the height of the patella ridge and minimize shear forces on the repair tissue.[96] In a study of 11 patients treated with this technique, Lysholm scores were 75 compared with 60 for the control group.

Realignment Procedures

Realignment procedures have long been known to affect the outcome of cartilage lesions in the PF joint. In 1997, Pidoriano *et al*.[97] correlated the results of AMZ to the location of PF chondral lesions. Of the 36 patients, 57% had at least 1 PF dislocation. They found poorer results in those with medial and diffuse lesions (Fulkerson type III and IVB). A recent systematic review demonstrated a greater improvement in outcome measures for patients undergoing combined ACI and osteotomy compared with ACI alone.[98] In 2006, Henderson and Lavigne[74] reported on 44 patients with either patella ACI or patella ACI with medializing tibial tuberosity osteotomy based on unspecified clinical and radiological indicators of malalignment. At an average of 2 years of follow-up, the combined group had better modified Cincinnati knee scores (4.5 vs. 1.7 points), better SF-36 physical component scores (70.9 vs. 55.4 points), and higher IKDC scores (85.2 vs. 60.6 points) compared with the ACI only group. They felt that malalignment may have been underdiagnosed in the ACI only group, and that a realignment procedure may be desirable in some patients even with normal PF tracking. Gigante *et al*.[73] reported on 14 knees treated with matrix-seeded ACI and AMZ. Malalignment was defined as TT-TG >20 mm on CT scans. Average Lysholm score improved from 55 to 92.5, and Tegner Activity Scale from 1 to 4. Pascual-Garrida *et al*.[93] reported the results of PF ACI in 52 patients, 28 of whom had simultaneous AMZ. Exact indications for AMZ were not specified. Patients undergoing AMZ showed a greater improvement in Lysholm scores (33 to 67 vs. 31 to 57) and better final IKDC scores (63 vs. 46) at average 4 years' follow-up. Interestingly, those with a previous failed microfracture had the highest average final Lysholm score of 70. There are no studies into the effect of tibial tuberosity distalisation or MPFL reconstruction on the result of cartilage surgery in the PF joint.

The improvement seen with realignment may not be without risk. Vasiliadis *et al*.[99] reported the results of 92 patients treated with ACI for PF lesions at a mean follow-up of 12.6 years. In this retrospective study, 38 patients had some type of realignment procedure. Results in the 2 groups were similar (Lysholm score 70.5 and Tegner 3 with realignment, 70 and 3 ACI only); however, the rate of serious complications was 29% in the realignment group compared with 13% in the ACI only group. It is also unclear if the benefit of combined ACI and realignment is also observed in the predominantly medial sided lesions seen in acute PF dislocation, particularly as increased medial degenerative change has been reported after realignment procedures.[35]

Arthroplasty

In advanced PF arthrosis, particularly in cases of trochlear dysplasia, arthroplasty may be an appropriate salvage

procedure.[100] A number of PF prostheses are available, with inlay and onlay designs. There is some controversy over the choice between PF arthroplasy and total knee arthroplasty in isolated PF arthrosis, particularly in older patients.

Future Directions

An improved understanding of the mechanisms of cell death and matrix degradation in acute chondral injury may afford the opportunity for early, nonsurgical intervention to limit cartilage damage.[101-103] A number of studies in animal models[104-106] and human cartilage[107,108] have demonstrated reduced cell death and cartilage degradation with the use of agents such as P188 surfactant, bone morphogenic proteins (BMP-7), *N*-acetylcysteine, and capsase inhibitors.

Conclusion

Patellofemoral instability is common and affects a predominantly young age group. Acute chondral injury frequently occurs, most commonly affecting the inferomedial patella. Biomechanical abnormalities, such as trochlear dysplasia, patella alta, and increased tibial tuberosity-trochlear groove distance, predispose to both recurrent dislocations and patellofemoral arthrosis.[32]

Given the increased severity of chondral lesions and progression to OA seen with recurrent dislocations,[60,61] careful consideration should be given to early stabilisation in patients with these predisposing factors. Although older stabilisation procedures were associated with increased rates of OA, recent evidence suggests that this is not the case with newer, more anatomical techniques.[39]

In general, chondral repair and restoration techniques demonstrate inferior outcomes in the PF joint compared with other compartments of the knee. Little evidence exists regarding the outcomes specifically in instability syndromes. Results of combined extensor mechanism realignment by AMZ and ACI are superior to ACI alone for patella lesions in malalignment; however, this may not hold true for the predominantly medial lesions seen after acute PF dislocation. The effects of MPFL reconstruction and tibial tuberosity distalization on PF cartilage procedures are unknown.

Improved understanding of the cellular mechanisms involved in chondral cell death and matrix degradation may allow early nonsurgical intervention to reduce cartilage damage after an acute injury.

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