Knee Surg Sports Traumatol Arthrosc (2016) 24:1836–1844
DOI 10.1007/s00167-016-4103-4

KNEE

Early osteoarthritis of the patellofemoral joint

Elizabeth A. Arendt[1] · Massimo Berruto[2] · Giuseppe Filardo[3] · Mario Ronga[4] · Stefano Zaffagnini[5] · Jack Farr[6] · Paolo Ferrua[2] · Alberto Grassi[5] · Vincenzo Condello[7]

Received: 15 December 2015 / Accepted: 22 March 2016 / Published online: 27 April 2016

Abstract Patellofemoral joint cartilage lesions are associated with a variety of clinical situations including blunt trauma, lateral patella dislocations, or as a secondary development in the setting of abnormal joint loading. There is a need for more clarity on how to best address these lesions. Most specifically, when is it necessary to surgically treat these lesions of the patella and trochlea and which technique to use? This review will focus on the spectrum of patellofemoral disease/injury and their treatment strategies, with special emphasis on cartilage damage and early osteoarthritis. Chapter sections will review the most common scenarios of cartilage damage in the patellofemoral joint, with an attempt to summarize current treatment, their outcomes, remaining challenges and unanswered questions.

Level of evidence V.

Keywords Osteoarthritis · Patellofemoral joint · Trochlea · Cartilage · Treatment

Introduction

The patellofemoral (PF) joint is composed of two osseous components, the patella and the femoral trochlea, which share a cartilaginous articulating surface. The patella is a sesamoid bone enclosed within the extensor mechanism; its function is intimately associated with dynamic lower limb muscle activity. From a neuromuscular viewpoint, the quadriceps muscle unit is responsible for knee extension in open kinetic chain function and

✉ Elizabeth A. Arendt
arend001@umn.edu

Massimo Berruto
massimo.berruto@gpini.it

Giuseppe Filardo
giuseppe@filardo.eu

Mario Ronga
mario.ronga@uninsubria.it

Stefano Zaffagnini
stefano.zaffagnini@unibo.it

Jack Farr
indyknee999@gmail.com

Paolo Ferrua
paolo.ferrua81@gmail.com

Alberto Grassi
alberto.grassi@ior.it

Vincenzo Condello
vincenzo.condello@sacrocuore.it

[1] Department of Orthopaedic Surgery, University of Minnesota, 2450 Riverside Av. So., Ste. R200, Minneapolis, MN 55454, USA

[2] Department of Knee Surgery, Orthopaedic Hospital Gaetano Pini, Milan, Italy

[3] Biomechanics Lab – II Clinic, Rizzoli Orthopaedic Institute, Bologna, Italy

[4] Orthopaedics and Traumatology, Department of Biotechnology and Life Sciences (DBSV), University of Insubria, Varese, Italy

[5] Istituto Ortopedico Rizzoli, Via di Barbiano 1/10, 40136 Bologna, Italy

[6] Indiana School of Medicine, Greenwood, IN, USA

[7] Sacro Cuore Hospital, Negrar, Verona, Italy

stabilization and shock absorption of the limb in closed kinetic chain activities. This complex orchestration of bony, soft tissue, cartilage and neuromuscular elements offers challenges to the clinician when faced with PF disease and injury.

The PF joint, due to its highly complex structure and function, is often affected by articular surface damage. PF cartilage damage has been documented in a large percentage of knee arthroscopy procedures, with the involvement of the articular surface of the PF joint in up to 44.6 % of these lesions [64]. Patellofemoral joint cartilage lesions may arise following blunt trauma, lateral patella dislocations, or as a secondary development in the setting of abnormal joint loading. Left untreated cartilage defects affecting patella or trochlea surfaces can alter the normal distribution of weight-bearing forces and may predispose to the development of OA [18], thus supporting the need for more clarity on how to best address these lesions. Most specifically, when is it necessary to surgically treat these lesions and which technique to use? Unfortunately, cartilage lesions of the PF joint can be particularly challenging to treat due to the complex biomechanical environment including varying individual anatomy, and the unique forces experienced within this compartment during weight-bearing and bent knee activities.

This review will discuss the spectrum of PF disease/injury and their treatment strategies, with special emphasis on cartilage damage and early osteoarthritis (OA). Chapter sections will review the most common scenarios of cartilage damage in the PF joint, with an attempt to summarize current treatment, their outcomes, remaining challenges and unanswered questions.

Post-traumatic PF lesions

The pathogenesis of PF cartilage lesions is frequently multifactorial; however, it is possible to identify a group of lesions with a traumatic aetiology. One frequent traumatic mechanism is blunt trauma, e.g. fall on a flexed knee, or a direct impact as seen in dashboard injuries. Often these cases show delayed chondral damage caused by the alteration of PF joint homoeostasis [54] or loss of articular congruence in the presence of a chondral/osteochondral lesion. The most common location of the cartilage lesion is a central bipolar lesion of both patella and trochlea, or the superomedial aspect of patella resulting from the frequent flexed knee position with this injury. Post-traumatic aetiology (including fractures, excluding patellar dislocations) accounted for 9 % of a large cohort of patients with isolated PF osteoarthritis [17].

PF chondral lesions related to patellar instability

Acute injuries

Osteochondral fractures are frequently observable after a patellar dislocation [51]; Nomura et al. [40] reported a 95 % incidence of cartilage damage with 72 % of osteochondral lesions, mostly located on medial patellar facet. MR imaging is crucial in assessing the dimension and location of the damaged articular surface, though arthroscopic examination allows better evaluation of the involved articular surface and allows definitive treatment of small focal defects. With a viable osteochondral lesion involving more than 10 % of the PF joint articular surface, acute surgical repair is advised [9]. Osteochondral fragments should be fixed in place using resorbable pins or headless compression screws in the acute phase to preserve chondrocytes viability and restore PF articular congruency. When chondral or osteochondral lesions cannot be repaired, persistency of pain and swelling may make a delayed chondral surgical treatment necessary. Potential cartilage techniques used are discussed later in this section.

Recurrent patellar instability

Grelsamer et al. [17] reported a history of patellar dislocation in 33 % of isolated PF OA, with PF instability the most common identifiable cause of this pathology. Vollnberg et al. [62] showed a strong correlation between the number of patellar dislocation and the prevalence of PF OA assessed by MRI, confirming similar (arthroscopic) observations of Nomura and Inoue [38].

The association of the anatomic factors with recurrent patellar instability has been established [7]; these being trochlear dysplasia (TD), patella alta, excessive tibial tubercle–trochlear groove (TT–TG) distance and excessive lateral patellar tilt. However, these same factors are found in a large cohort of isolated patella OA patients [17] (Table 1). This presents a confusing picture of the aetiology of PF OA in patients with patellar instability.

Although cartilage procedures can be performed in association with patellar stabilization surgery [32], there is no consensus on this topic and sparse literature support for this combined procedure. This is due, in part, to the generally good results of stabilization surgery and mixed results of cartilage restoration techniques in this joint (as discussed later in this section).

Siebold et al. [47] reported the results of a ten patients presenting an ICRS grade 4 patellar lesions in association with recurrent patellar dislocation; surgery performed was an MPFL reconstruction plus autologous chondrocyte implantation (ACI). At a mean follow-up of 2 years, no

Table 1 The % PF anatomic factors associated with PF OA/instability

	Patella alta	Trochlear dysplasia	Excessive patella tilt	Excessive TT–TG
PF OA	No statistical threshold	78 %	66 %	No statistical threshold
PF instability (%)	30	96	83	56

PF patellofemoral, *OA* osteoarthritis, *TT–TG* tibial tubercle–trochlear groove

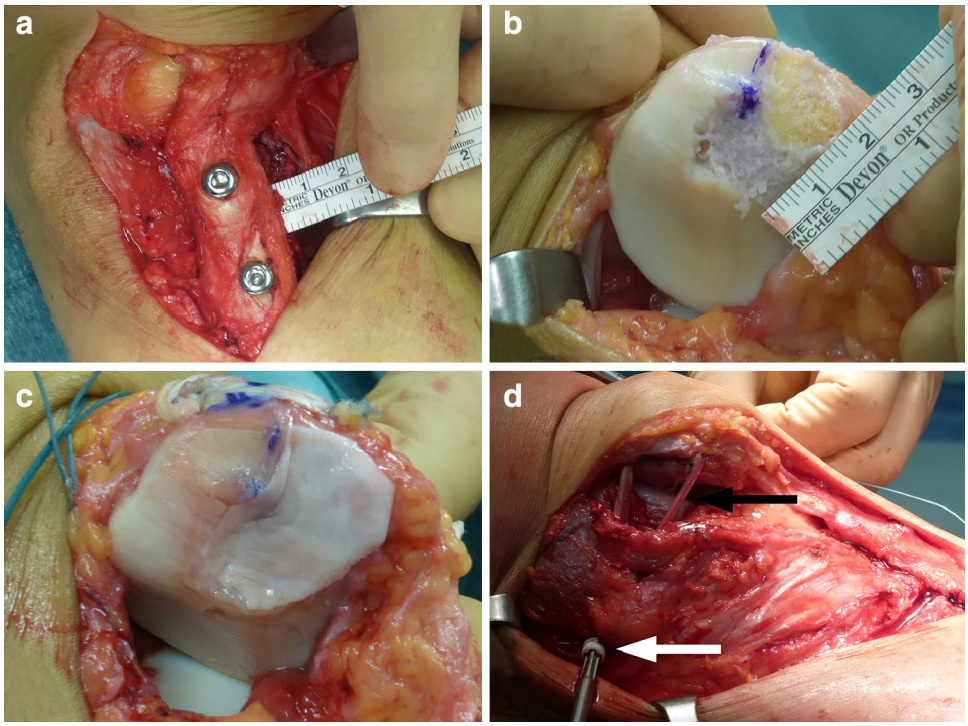

Fig. 1 Thirty-three year-old women suffering from recurrent patella dislocation and chondromalacia of medial patella facet. **a** Emslie-Trillat procedure with a medialization of tibial tuberosity of 8 mm. **b** After debridement, the chondral lesion measured 4.5 cm^2. **c** AMIC (Autologous Matrix-Induced Chondrogenesis). **d** MPFL reconstruction with autologous gracilis tendon (*black arrow*) fixed on patella with 2 anchor and on femur with an interference screw (*white arrow*). This figure was generously provided by Dr. Mario Ronga

recurrence of patellar dislocation was observed. Subjective and objective scores were increased over pre-op scores; however, the improvement was not statistically significant. This short-term study questions the value of a combined cartilage surgery to optimize the final outcome.

Without evidence-based literature on the topic of when to treat cartilage lesions concurrent with patellar stabilization surgery, most surgeons limit surgical treatment to patellar stabilization alone. Treatment of chondral lesions associated with PF instability need to be performed when, after a surgical patellar stabilization, the damage remains symptomatic as judged by pain and recurrent swelling (Fig. 1). Also, after a failed patellar surgical stabilization procedure, cartilage lesions are often addressed concomitant with revision surgery, when present.

Patellar cartilage lesions without patellar dislocations

Dejour et al. [7] defined potential patella instability (PPI) patients as those patients that have (+) anatomic instability factors without patellar dislocations; PF cartilage damage is frequently observed in these patients. High-grade (B, C, D) TD was found in 78 % of 365 patients with isolated PF OA, validating that PF OA is associated with TD with or without a history of patellar instability [17]. Within this cohort, only 33 % had a history of patellar instability. In a study of patients undergoing anteromedialization of the tibial tubercle for symptomatic PF cartilage lesions, only 58 % of patients reported a history of patella dislocations [44]. These studies support the association of PF cartilage lesions without patella instability.

The aetiology of cartilage wear is speculative. In the presence of a trochlear bump, as observed in TD type B and D, or in the presence of a trochlear prominence (TD type C), the prominence of the proximal trochlea raises the contact pressures on PF joint, causing an "anti-Maquet effect" during the first degrees of flexion [30, 59]. In the setting of patella alta, excessive load of the distal patella can occur due to decreased engagement of the patella in the trochlea, concentrating load on a smaller than normal area of cartilage with a resulting increase in cartilage load, which may

result in cartilage wear [25]. Excessive TT–TG distance and/or excessive patellar tilt can increase lateral patella facet pressure with eventual lateral patellar shift, predisposing the lateral PF joint to increased load and possible cartilage damage [58]. Excessive TT–TG distance presents a direct correlation with axial malalignment and subsequent lateral facet cartilage damage [52]; indeed, TT–TG values have been shown to be proportional to the development of PF cartilage damage and OA [15].

Symptomatic defects of the patella can improve following isolated realignment surgery. The *location* of the cartilage lesion correlates better with good outcome after anteromedialization (AMZ) procedure (Fig. 2a, b) than the *depth and extent* of the lesion in a nonhomogeneous population with either lateral patella subluxation or symptomatic PF OA [44]; patients undergoing AMZ had better outcomes when lesions were located in the lateral facet or distal pole of the patella. Patients had poor outcomes with medial and proximal patellar lesions. The central trochlear lesion is a contraindication to the AMZ since all such lesions resulted in poor outcomes. Conversely, all lateral trochlear lesions, most of them associated with lateral patellar lesions, had good or excellent results. The conclusion of this study was that normal knee function after this procedure may not occur, but the patients may experience improvement in pain, stability and activities of daily living [44].

In potential patella instability [7] patients with symptomatic chondral lesions, surgery aimed at the correction of predisposing instability factors (realignment surgery) and reparative cartilage surgery has some literature support

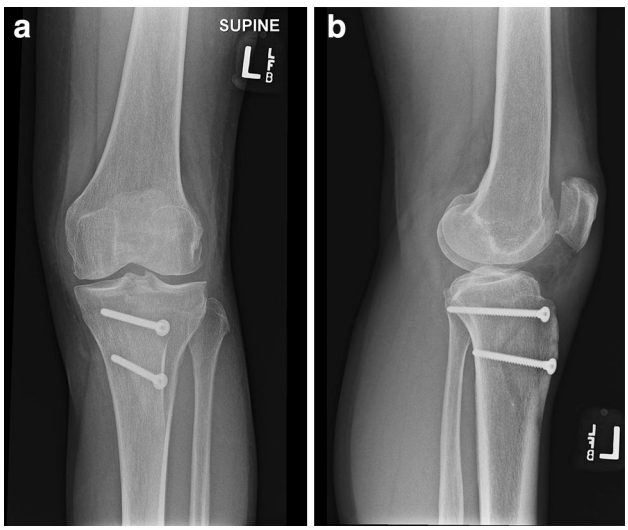

Fig. 2 **a** AP knee radiograph of a knee with cartilage restoration combined with an anteromedialization (AMZ) realignment procedure. **b** Lateral knee radiograph of a knee with cartilage restoration combined with an anteromedialization (AMZ) realignment procedure. Figures were generously provided by Dr. Andreas Gomoll

for this combined approach. The aim of these procedures is to unload the cartilage repair area and restore a physiologic tracking of the PF joint, with the goal of relieving pain and optimizing cartilage restoration potential. In a population presenting a symptomatic patellar cartilage lesion in association with a predisposing factor (TT–TG distance >20 mm), Gigante et al. [14] found significant improvement of clinical scores (Kujala from 52 to 88.5) at midterm follow-up after combined distal realignment and matrix-assisted autologous chondrocyte transplantation (MACT). In most current published studies, the association of 'realignment procedures' improves the outcomes of cartilage procedures as evidenced by the decrease in pain [10, 43, 60], though with increased complications [61]. A recent systematic review [57] showed a significant increase in clinical results at a midterm follow-up in patients treated with associated realignment procedures compared to isolated ACI. Though there is general acceptance to correct predisposing anatomic factors simultaneous with cartilage restoration surgery in patients without a history of patellar dislocation, there remains no consensus on the amount of correction needed in order to avoid possible complications related to hypercorrection, such as patella infera or hypermedialization.

The inferior results observed in central patellar and trochlear defects lie in the specific biomechanical properties of the PF compartment of the knee joint [56]. During walking or stair climbing, the patellar ridge has a significant meshing function in guiding the patella in the sliding bearing and prevents gliding movements in the coronary plane [56]. The patella sustains forces in excess of seven times body weight, with shear forces that are greater than those in the tibiofemoral knee compartments. Thus, full thickness patellar ridge chondral lesions or flattening due to excessive cartilage debridement is associated with poorer outcomes [31, 37]. For this reasons, many authors have suggested preserving this anatomic region, i.e. the median ridge, when possible [31, 37]. Niemeyer et al. [36] have described the "double eye technique": this technique provides for a separate reconstruction of the medial and the lateral facets by means of ACI, but the median ridge region is preserved to maintain the original thickness of cartilage at this site. The authors [37] reported better results in patients treated with ACI and preservation of the median patellar ridge compared with patients treated with ACI with excessive debridement and flattening the this ridge.

There is a need for higher-level studies to give systematic recommendations for treating or not treating cartilage damages as concomitant procedure in surgical correction of patellar instability. In regard to treating all concomitant instability factors, there remain questions of when and how much correction is needed.

Does surgical treatment of patellar instability prevent early OA?

Whether PF realignment surgery is able to prevent or delay PF early OA is a much debated but still unsolved issue. Published literature on surgical solutions for PF instability contains diverse surgical methods, as well as diverse methods of recording pre- and post-operative demographic and imaging variables [55]. The majority of papers report populations with mixed diagnoses and inconsistent applicability to the broad spectrum of patellar instability patients. The outcomes in the current literature need more clarity and consistency in reporting methodology to be of value for the treating clinician [55].

Due to the multifactorial nature of PF instability, the spectrum of varying anatomic abnormalities, and diverse surgical solutions, the current literature is scarce of high-quality studies regarding the long-term natural history of lateral patellar dislocation and the effect of surgical correction.

In 1992, Arnbjornsson et al. [1] published a seminal study addressing this concern; the authors evaluated patients with bilateral recurrent PF instability treated surgically in one knee and conservatively in the contralateral knee, demonstrating comparable results at the short term. However, at a mean of 14-year follow-up, 75 % of the operated knees presented with OA degenerative changes compared to 29 % in the conservatively managed knees. Study limitations included: small cohort, unclear OA grading system and heterogeneous (and at times outdated) surgical management.

A similar study design was applied by Marcacci et al. [28], who reported on 16 patients with a mean 30-year follow-up; there were similar clinical and radiographic results reported on patients with the Roux surgical technique compared to conservative management. Despite marked degenerative changes in 50 % of both knees, there was a greater percentage of conservatively treated knees with a lower grade OA (50 vs. 31 %). This study presents similar limitations, small cohort and the use of a dated non-anatomic surgical procedure.

There are important factors to consider when interpreting the findings of these studies. There is a natural development of PF OA in knees with PF instability. This was also confirmed by Maenpaa and Lehto [26] who reported PF OA in 22 % of patellar instability knees compared to 11 % of contralateral healthy knees at 13-year follow-up. Another consideration is the apparent inability of PF stabilization surgery to prevent or delay PF OA onset and progression. All of the surgical procedures discussed in these articles were based on the principle of extensor mechanism realignment as the key factor in patellar stabilization, most involving a medialization of the tibial tubercle and/or distal patella tendon. The effect of this kind of surgery appears to worsen the radiographic results, possibly due to the changes in PF kinematics and pressures caused by the realignment procedures [24, 27], or incomplete or inadequate correction of PF abnormalities.

Current techniques addressing patella containment, i.e. MPFL reconstruction, combined with less post-op immobilization and a more knowledgeable approach to post-surgical rehabilitation, could translate into surgical management with more encouraging long-term results. This is suggested by a few short- and midterm studies. Sillanpaa et al. [48, 49] reported on two different surgical techniques with different results in terms of OA progression. The authors reported no OA signs and better radiographic results at 10-year follow-up in patients treated with MPFL reconstruction through adductor magnus tenodesis compared to the non-anatomic Roux surgical technique. Nomura et al. [39] reported (only) 12 % of moderate PF OA 12 years after MPFL reconstruction. More discouraging is a study from Farr et al. [11] who studied 30 knees in 26 patients who underwent medialization of the tibial tubercle for recurrent patella instability. This study was a case–control study with a minimum 10-year follow-up reporting severe PF and tibiofemoral OA in 23 % of patients. However, radiographic findings and subjective clinical reporting were not statistically different than the controls. The authors also reported worst radiographic results in patients treated late, opining that delay in surgical treatment, allowing recurrent patellar dislocations, could be responsible for further chondral damage, as suggested by others [33, 34].

A procedure used to surgically treat patella instability combined with high-grade dysplasia is a trochleoplasty [3, 5, 42]. A recent systematic review [50] including six studies showed higher-grade PF OA in patients with severe TD (Dejour type B–D) treated with non-trochleoplasty procedures (Insall's proximal realignment) [46] compared to trochleoplasty [2, 6, 41, 53, 63]. Failing to address TD may produce poor radiographic results; alternatively, severely dysplastic groove abnormalities requiring trochleoplasty are correlated with both worst clinical and radiographic outcomes compared to less severe presentations where realignment procedures may suffice [29, 65]. Other considerations for OA associated with trochleoplasties include: the presence of high degenerative changes at time of surgery in severe TD [6, 41, 63], and PF incongruence due to a dysplastic patella tracking in a newly deepened trochlear groove [45].

There is no current evidence that suggests surgical treatment of patellar instability prevents or delays early PF OA. Despite the potential for future OA, even after surgical stabilization procedures, positive clinical results in terms of symptom reduction and prevention of recurrence should

guide surgical management. There are short-term studies concerning patella stabilization currently in our literature; it will take appropriate study designs detailing cartilage damage at the time of the surgery, combined with surgical outcomes and long-term follow-up, to provide the necessary information to answer whether patella stabilization surgery, with or without combined cartilage procedures, has a positive or negative impact on OA prevention or progression.

Overview of cartilage treatment of the PF joint

The surgical procedures adopted for the treatment of cartilage defects of the PF joint have been borrowed from the techniques successfully used for femoral condyles lesions, even though with sometimes less satisfactory clinical results [23].

Arthroscopic chondral debridement, focused on removing fibrillated cartilage to smooth a rough articular surface, has been applied with encouraging positive subjective results [12]. However, microtraumatic/degenerative lesions performed worse than traumatic cases, and many patients who were improved by the surgery still presented functional limitations. Currently, its use is rarely indicated as a primary procedure.

Microfracture, a bone marrow stimulation technique, proved to be effective in improving symptoms and function at short-term follow-up [22], and it is usually the first choice procedure to manage small cartilage lesions. However, patients with lesions in the PF joint obtained lower results at all follow-ups times compared to patients with condylar lesions, and an overall progressive worsening of the evaluated scores has been shown over time [22].

Some efforts have been made in the recent years in order to improve microfracture healing potential through augmentation with biomaterials, which should allow a better reparative tissue and thus greater improvement in subjective scores and longer-lasting results. Autologous matrix-induced chondrogenesis (AMIC), which combines microfracturing with a collagen I/III matrix, demonstrated a satisfying clinical improvement at 24-month follow-up for the treatment of PF cartilage defects in the knee. However, the favourable clinical outcome of the AMIC technique was not confirmed by the magnetic resonance image (MRI) findings, with frequent subchondral lamina changes and intra-lesional osteophytes [8].

For those lesions characterized by greater cartilage loss, different treatment approaches have been proposed.

Autologous osteochondral transplantation or mosaicplasty, which provide a single-stage procedure with immediate reliable tissue transfer of a viable osteochondral unit capitalizing on bone-to-bone healing, has been used in the PF joint with controversial results: an overall improvement in clinical scores but with a high failure rate [4]. The reason may be the difficulty in accurately reproducing the curvature of the articular surface of both the patella and the trochlea. Moreover, osteochondral plugs harvested from a low load-bearing location in the femoral trochlea have a thinner cartilage layer compared to surrounding patellar tissue, resulting in a discrepancy at the osteochondral interface when performing this technique on the patella. Allograft osteochondral transplantation does not suffer from such limitations because the donor graft is harvested from the same location and is size matched. Nonetheless, a high failure rate was shown at long-term follow-up, which was felt to be related to anatomic considerations, including residual dysplasia, functional defects of the limb or knee, combined with the difficulties in restoring the peculiar anatomy of this region [16].

First-generation ACI, based on two surgical steps for chondrocyte harvesting, culture, and subsequent implantation into the defect site, has been proven as a suitable option for cartilage lesions in the PF joint [31]. However, the long-term evaluation for this regenerative approach also provided inferior results for degenerative lesions and a higher failure rate for patients affected by patellar (vs. knee) defects [35]. MACT procedures have been introduced to overcome some of the limitations of the first-generation approach, involving the growth of patients' chondrocytes on three-dimensional scaffolds of various biocompatible materials. A long-term study on the use of a hyaluronan-based MACT showed significant clinical improvement for both patellar and trochlear lesions with a low number of failures in follow-up of up to 10 years; however, significantly lower results were found in complex cases, e.g. female patients with patellar lesions requiring realignment procedures [21].

Osteochondral scaffolds represent the last frontier for the regeneration of the articular surface, an "off-the-shelf" approach with different biomaterials designed to replace the entire damaged osteochondral unit in a single-step procedure. A polylactic, polyglycolic acid and calcium phosphate scaffold were used with very unsatisfactory results in the PF joint: poor clinical scores after 2 years, a failure rate of 70 % and cylindrical cavity of fibrous tissue instead of subchondral bone restoration at MRI [19]. Better results have recently been reported for another scaffold made of three layers of type I collagen and hydroxyapatite in different concentrations to reproduce the structure and composition of the osteochondral unit: good clinical scores were found at the midterm follow-up, with evidence of a slow but progressive maturation of the scaffold at MRI evaluation, even though the persistence of some abnormalities suggested some limitations in this technique. There remains the possibility to further improve this surgical approach for one-step treatment of chondral and osteochondral lesions [20].

Several treatment options have been proposed to treat cartilage lesions in the PF joint, but none have emerged as gold standard, neither to improve symptoms and function nor to prevent OA degeneration. One of the reasons for the overall unsatisfactory results may be due to the poor understanding of the pathology itself and therefore the inclusions of heterogeneous pathological entities in the same study cohorts, a bias leading to inconclusive findings. In this regard, it has recently been shown how the same cartilage surgical technique used in the PF compartment led to different results, with patellar lesions demonstrating lower results compared to trochlear lesions [13]. This suggests that the PF joint articulating surfaces may have different lesion patterns (aetiologies) as well as different healing mechanisms. In future cartilage studies involving the PF joint, lesions should be separated by location and evaluated separately, to better understand the potential for cartilage treatments in this region. Moreover, the literature lacks properly designed studies with agreement on the diagnosis of early OA, indications of the available techniques in the early OA phase, and consistency with reporting data and outcomes. There is a need for high-level studies to establish the value of cartilage treatments in early OA, but also to understand the aetiopathogenetic processes and the biomechanical alterations responsible for these degenerative lesions and better target available and advancing treatment options.

Conclusion

Despite increasing interest and literature publications in these topics, questions remain:

Is the location of the cartilage lesion in the PF joint important in the development of clinical symptoms and/or eventual OA?
Which cartilage treatment is best for each location (including non-operative solutions)?
When is it advisable to combine a cartilage procedure with patella stabilization surgery in patella instability patients?
When is it necessary to combine realignment surgery in PPI patients undergoing cartilage restoration surgery?
Does patellar stabilization change the natural history of patellofemoral OA associated with trochlear dysplasia and other anatomic instability factors?
Does patella stabilization surgery have a positive or negative impact on OA prevention or progression?

The recurrent theme with each topic within this chapter is the need for higher-level studies to give systematic recommendations for treatment. For this, we need consensus in PF language, agreement in the clinical and imaging factors most important to record, and agreement in assessment tools for outcome evaluation.

References

1. Arnbjornsson A, Egund N, Rydling O (1992) The natural history of recurrent dislocation of the patella: long-term results of conservative and operative treatment. J Bone Joint Surg (Br) 74:140–142
2. Banke IJ, Kohn LM, Meidinger G, Otto A, Hensler D, Beitzel K, Imhoff AB, Schottle PB (2014) Combined trochleoplasty and MPFL reconstruction for treatment of chronic patellofemoral instability: a prospective minimum 2-year follow-up study. Knee Surg Sports Traumatol Arthrosc 22:2591–2598
3. Beaufils P, Thaunat M, Pujol N, Scheffler S, Rossi R, Carmont M (2012) Trochleoplasty in major trochlear dysplasia: current concepts. Sports Med Arthrosc Rehabil Ther Technol 4:7
4. Bentley G, Biant L, Carrington R, Akmal M, Goldberg A, Williams A, Skinner J, Pringle J (2003) A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. J Bone Joint Surg (Br) 85-B:223–230
5. Bremer Hinckel B, Arendt EA, Ntagiopoulos PG, Dejour D (2016) Trochleoplasty: historical overview and Dejour technique. Oper Tech Sports Med 23:114–122
6. Dejour D, Byn P, Ntagiopoulos PG (2013) The Lyon's sulcus-deepening trochleoplasty in previous unsuccessful patellofemoral surgery. Int Orthop 37:433–439
7. Dejour H, Walch G, Nove-Josserand L, Guier C (1994) Factors of patellar instability: an anatomic radiographic study. Knee Surg Sports Traumatol Arthrosc 2:19–26
8. Dhollander A, Moens K, Van der Maas J, Verdonk P, Almqvist KF, Victor J (2014) Treatment of patellofemoral cartilage defects in the knee by autologous matrix-induced chondrogenesis (AMIC). Acta Orthop Belg 80:251–259
9. Duthon VB (2015) Acute traumatic patellar dislocation. Orthop Traumatol Surg Res 101:S59–S67
10. Farr J 2nd (2008) Autologous chondrocyte implantation and anteromedialization in the treatment of patellofemoral chondrosis. Orthop Clin North Am 39:329–335
11. Farr S, Huyer D, Sadoghi P, Kaipel M, Grill F, Ganger R (2014) Prevalence of osteoarthritis and clinical results after the Elmslie–Trillat procedure: a retrospective long-term follow-up. Int Orthop 38:61–66
12. Federico DJ, Reider B (1997) Results of isolated patellar debridement for patellofemoral pain in patients with normal patellar alignment. Am J Sports Med 25:663–669
13. Filardo G, Kon E, Andriolo L, Di Martino A, Zaffagnini S, Marcacci M (2014) Treatment of "patellofemoral"cartilage lesions with matrix-assisted autologous chondrocyte transplantation: a comparison of patellar and trochlear lesions. Am J Sports Med 42:626–634
14. Gigante A, Enea D, Greco F, Bait C, Denti M, Schonhuber H, Volpi P (2009) Distal realignment and patellar autologous chondrocyte implantation: mid-term results in a selected population. Knee Surg Sports Traumatol Arthrosc 17:2–10
15. Goutallier D, Bernageau J, Lecudonnec B (1978) The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). Rev Chir Orthop Reparatrice Appar Mot 64:423–428
16. Gracitelli GC, Meric G, Pulido PA, Gortz S, De Young AJ, Bugbee WD (2015) Fresh osteochondral allograft transplantation for isolated patellar cartilage injury. Am J Sports Med 43:879–884

17. Grelsamer RP, Dejour D, Gould J (2008) The pathophysiology of patellofemoral arthritis. Orthop Clin North Am 39:269–274
18. Insall J, Falvo KA, Wise DW (1976) Chondromalacia patellae. A prospective study. J Bone Joint Surg Am 58:1–8
19. Joshi N, Reverte-Vinaixa M, Diaz-Ferreiro EW, Dominguez-Oronoz R (2012) Synthetic resorbable scaffolds for the treatment of isolated patellofemoral cartilage defects in young patients: magnetic resonance imaging and clinical evaluation. Am J Sports Med 40:1289–1295
20. Kon E, Filardo G, Di Martino A, Busacca M, Moio A, Perdisa F, Marcacci M (2014) Clinical results and MRI evolution of a nano-composite multilayered biomaterial for osteochondral regeneration at 5 years. Am J Sports Med 42:158–165
21. Kon E, Filardo G, Gobbi A et al (2015) Long-term results after hyaluronan-based MACT for the treatment of cartilage lesions of the patello-femoral joint (submitted)
22. Kreuz PC, Steinwachs MR, Erggelet C, Krause SJ, Konrad G, Uhl M, Sudkamp N (2006) Results after microfracture of full-thickness chondral defects in different compartments in the knee. Osteoarthr Cartil 14:1119–1125
23. Krishnan SP, Skinner JA, Bartlett W, Carrington RW, Flanagan AM, Briggs TW, Bentley G (2006) Who is the ideal candidate for autologous chondrocyte implantation? J Bone Joint Surg Br 88:61–64
24. Kuroda R, Kambic H, Valdevit A, Andrish J (2001) Articular cartilage contact pressure after tibial tuberosity transfer. A cadaveric study. Am J Sports Med 29:403–409
25. Lording T, Lustig S, Servien E, Neyret P (2014) Chondral injury in patellofemoral instability. Cartilage 5:136–144
26. Maenpaa H, Lehto MU (1997) Patellofemoral osteoarthritis after patellar dislocation. Clin Orthop Relat Res 339:156–162
27. Mani S, Kirkpatrick MS, Saranathan A, Smith LG, Cosgarea AJ, Elias JJ (2011) Tibial tuberosity osteotomy for patellofemoral realignment alters tibiofemoral kinematics. Am J Sports Med 39:1024–1031
28. Marcacci M, Zaffagnini S, Iacono F, Visani A, Petitto A, Neri NP (1995) Results in the treatment of recurrent dislocation of the patella after 30 years' follow-up. Knee Surg Sports Traumatol Arthrosc 3:163–166
29. Marcacci M, Zaffagnini S, Lo Presti M, Vascellari A, Iacono F, Russo A (2004) Treatment of chronic patellar dislocation with a modified Elmslie–Trillat procedure. Arch Orthop Trauma Surg 124:250–257
30. Mehl J, Feucht MJ, Bode G, Dovi-Akue D, Sudkamp NP, Niemeyer P (2014) Association between patellar cartilage defects and patellofemoral geometry: a matched-pair MRI comparison of patients with and without isolated patellar cartilage defects. Knee Surg Sports Traumatol Arthrosc 24:838–846
31. Minas T, Bryant T (2005) The role of autologous chondrocyte implantation in the patellofemoral joint. Clin Orthop Relat Res 436:30–39
32. Mouzopoulos G, Borbon C, Siebold R (2011) Patellar chondral defects: a review of a challenging entity. Knee Surg Sports Traumatol Arthrosc 19:1990–2001
33. Nakagawa K, Wada Y, Minamide M, Tsuchiya A, Moriya H (2002) Deterioration of long-term clinical results after the Elmslie–Trillat procedure for dislocation of the patella. J Bone Joint Surg (Am) 84-B:861–864
34. Naveed MA, Ackroyd CE, Porteous AJ (2013) Long-term (ten- to 15-year) outcome of arthroscopically assisted Elmslie–Trillat tibial tubercle osteotomy. Bone Joint J 95-B:478–485
35. Nawaz SZ, Bentley G, Briggs TW, Carrington RW, Skinner JA, Gallagher KR, Dhinsa BS (2014) Autologous chondrocyte implantation in the knee: mid-term to long-term results. J Bone Joint Surg Am 96:824–830
36. Niemeyer P, Kreuz PC, Steinwachs M, Kostler W, Mehlhorn A, Kraft N, Sudkamp NP (2007) Technical note: the "double eye" technique as a modification of autologous chondrocyte implantation for the treatment of retropatellar cartilage defects. Knee Surg Sports Traumatol Arthrosc 15:1461–1468
37. Niemeyer P, Steinwachs M, Erggelet C, Kreuz PC, Kraft N, Kostler W, Mehlhorn A, Sudkamp NP (2008) Autologous chondrocyte implantation for the treatment of retropatellar cartilage defects: clinical results referred to defect localisation. Arch Orthop Trauma Surg 128:1223–1231
38. Nomura E, Inoue M (2005) Second-look arthroscopy of cartilage changes of the patellofemoral joint, especially the patella, following acute and recurrent patellar dislocation. Osteoarthr Cartil 13:1029–1036
39. Nomura E, Inoue M, Kobayashi S (2007) Bilateral recurrent patellar dislocation in a patient with isolated patella aplasia-hypoplasia. Arthroscopy 23(1136):e1131–e1134
40. Nomura E, Inoue M, Kurimura M (2003) Chondral and osteochondral injuries associated with acute patellar dislocation. Arthroscopy 19:717–721
41. Ntagiopoulos PG, Byn P, Dejour D (2013) Midterm results of comprehensive surgical reconstruction including sulcus-deepening trochleoplasty in recurrent patellar dislocations with high-grade trochlear dysplasia. Am J Sports Med 41:998–1004
42. Ntagiopoulos PG, Dejour D (2014) Current concepts on trochleoplasty procedures for the surgical treatment of trochlear dysplasia. Knee Surg Sports Traumatol Arthrosc 22:2531–2539
43. Pascual-Garrido C, Slabaugh MA, L'Heureux DR, Friel NA, Cole BJ (2009) Recommendations and treatment outcomes for patellofemoral articular cartilage defects with autologous chondrocyte implantation: prospective evaluation at average 4-year follow-up. Am J Sports Med 37(Suppl 1):33S–41S
44. Pidoriano AJ, Weinstein RN, Buuck DA, Fulkerson JP (1997) Correlation of patellar articular lesions with results from anteromedial tibial tubercle transfer. Am J Sports Med 25:533–537
45. Rouanet T, Gougeon F, Fayard JM, Remy F, Migaud H, Pasquier G (2015) Sulcus deepening trochleoplasty for patellofemoral instability: a series of 34 cases after 15 years postoperative follow-up. Orthop Traumatol Surg Res 101:443–447
46. Schuttler KF, Struewer J, Roessler PP, Gesslein M, Rominger MB, Ziring E, Efe T (2013) Patellofemoral osteoarthritis after insall's proximal realignment for recurrent patellar dislocation. Knee Surg Sports Traumatol Arthrosc 22:2623–2628
47. Siebold R, Karidakis G, Fernandez F (2014) Clinical outcome after medial patellofemoral ligament reconstruction and autologous chondrocyte implantation following recurrent patella dislocation. Knee Surg Sports Traumatol Arthrosc 22:2477–2483
48. Sillanpaa P, Mattila VM, Visuri T, Maenpaa H, Pihlajamaki H (2008) Ligament reconstruction versus distal realignment for patellar dislocation. Clin Orthop Relat Res 466:1475–1484
49. Sillanpaa PJ, Mattila VM, Visuri T, Maenpaa H, Pihlajamaki H (2011) Patellofemoral osteoarthritis in patients with operative treatment for patellar dislocation: a magnetic resonance-based analysis. Knee Surg Sports Traumatol Arthrosc 19:230–235
50. Song GY, Hong L, Zhang H, Zhang J, Li X, Li Y, Feng H (2014) Trochleoplasty versus nontrochleoplasty procedures in treating patellar instability caused by severe trochlear dysplasia. Arthroscopy 30:523–532
51. Stanitski CL, Paletta GA Jr (1998) Articular cartilage injury with acute patellar dislocation in adolescents. Arthroscopic and radiographic correlation. Am J Sports Med 26:52–55
52. Thakkar RS, Del Grande F, Wadhwa V, Chalian M, Andreisek G, Carrino JA, Eng J, Chhabra A (2015) Patellar instability: CT and MRI measurements and their correlation with internal derangement findings. Knee Surg Sports Traumatol Arthrosc. doi:10.1007/s00167-015-3614-8

53. Thaunat M, Bessiere C, Pujol N, Boisrenoult P, Beaufils P (2011) Recession wedge trochleoplasty as an additional procedure in the surgical treatment of patellar instability with major trochlear dysplasia: early results. Orthop Traumatol Surg Res 97:833–845
54. Thompson RC Jr, Oegema TR Jr, Lewis JL, Wallace L (1991) Osteoarthrotic changes after acute transarticular load. An animal model. J of Bone Joint Surg Ser A 73:990–1001
55. Tompkins MA, Arendt EA (2015) Patellar instability factors in isolated medial patellofemoral ligament reconstructions: what does the literature tell us? A systematic review. Am J Sports Med 43:2318–2327
56. Torzilli PA, Deng XH, Ramcharan M (2006) Effect of compressive strain on cell viability in statically loaded articular cartilage. Biomech Model Mechanobiol 5:123–132
57. Trinh TQ, Harris JD, Siston RA, Flanigan DC (2013) Improved outcomes with combined autologous chondrocyte implantation and patellofemoral osteotomy versus isolated autologous chondrocyte implantation. Arthroscopy 29:566–574
58. Tsavalas N, Katonis P, Karantanas AH (2012) Knee joint anterior malalignment and patellofemoral osteoarthritis: an MRI study. Eur Radiol 22:418–428
59. Van Haver A, De Roo K, De Beule M, Labey L, De Baets P, Dejour D, Claessens T, Verdonk P (2015) The effect of trochlear dysplasia on patellofemoral biomechanics: a cadaveric study with simulated trochlear deformities. Am J Sports Med 43:1354–1361
60. Vanlauwe JJ, Claes T, Van Assche D, Bellemans J, Luyten FP (2012) Characterized chondrocyte implantation in the patellofemoral joint: an up to 4-year follow-up of a prospective cohort of 38 patients. Am J Sports Med 40:1799–1807
61. Vasiliadis HS, Lindahl A, Georgoulis AD, Peterson L (2011) Malalignment and cartilage lesions in the patellofemoral joint treated with autologous chondrocyte implantation. Knee Surg Sports Traumatol Arthrosc 19:452–457
62. Vollnberg B, Koehlitz T, Jung T, Scheffler S, Hoburg A, Khandker D, Hamm B, Wiener E, Diederichs G (2012) Prevalence of cartilage lesions and early osteoarthritis in patients with patellar dislocation. Eur Radiol 22:2347–2356
63. von Knoch F, Bohm T, Burgi ML, von Knoch M, Bereiter H (2006) Trochleaplasty for recurrent patellar dislocation in association with trochlear dysplasia. A 4- to 14-year follow-up study. J Bone Joint Surg Br 88:1331–1335
64. Widuchowski W, Lukasik P, Kwiatkowski G, Faltus R, Szyluk K, Widuchowski J, Koczy B (2008) Isolated full thickness chondral injuries. Prevalence and outcome of treatment. A retrospective study of 5233 knee arthroscopies. Acta Chir Orthop Traumatol Cech 75:382–386
65. Zaffagnini S, Grassi A, Marcheggiani Muccioli GM, Luetzow WF, Vaccari V, Benzi A, Marcacci M (2014) Medial patellotibial ligament (MPTL) reconstruction for patellar instability. Knee Surg Sports Traumatol Arthrosc 22:2491–2498