

Chapter 4

Patellofemoral Arthritis



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Etiology of Patellofemoral Arthritis

The pathogenesis of wear and damage to articular cartilage of the knee is often multifactorial. However, with isolated patellofemoral (PF) arthritis (Fig. 4.1), the pathogenesis can most commonly be attributed to mechanical causes that overload the PF cartilage or traumatic shear injuries. These include trauma, patellar instability, malalignment, and dysplasia. Disruption of the articular surface leads to loss of the cartilage fluid pressure responsible for absorbing joint loads, and the resultant high stresses can lead to breakdown of collagen fibers and propagation of chondral defects. In non-traumatic cases, any aberrant mechanics of the PF articulation causes aberrant loads on the articular cartilage, predisposing the cartilage to breakdown over time.

Traumatic injury to the PF cartilage can occur either through blunt trauma (e.g., fall on a flexed knee or direct impact from a dashboard injury) or intraarticular fracture. The most common location of the cartilage lesion from this etiology is a central bipolar lesion of both the patella and trochlea or the superomedial aspect of the patella as a result of the knee being in a flexed position at the time of injury [1]. Posttraumatic etiologies account for approximately 9% of patients with isolated PF osteoarthritis (OA) [22].

Chondral and osteochondral shear injuries are frequently observed after a patellar dislocation, which predominately affects a younger population [44]. Patellar stability relies on limb alignment, the osseous containment of the patella within the trochlea, the integrity of the static and dynamic soft tissue constraints, and general-

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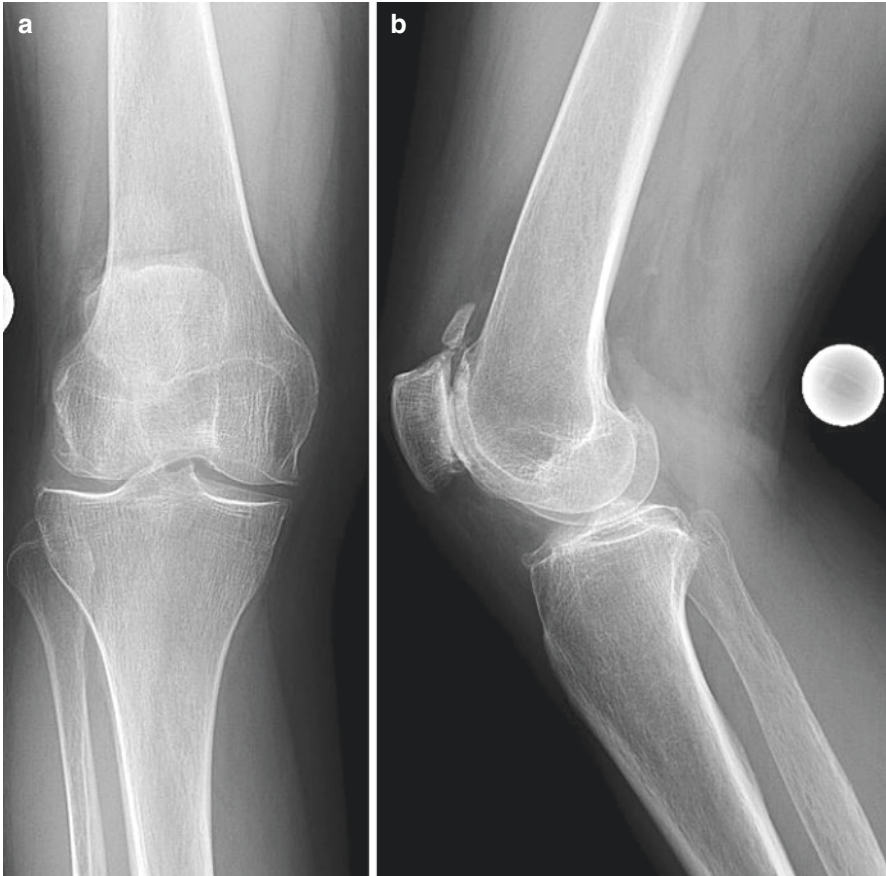


Fig. 4.1 (a) Anteroposterior and (b) lateral X-rays of a right knee with isolated patellofemoral arthritis

ized ligamentous laxity. Non-dysplastic knees require a high amount of energy to dislocate the patella from the trochlea, and therefore, the incidence of significant chondral or osteochondral injury to the medial patella facet in the setting of patellar dislocation can be up to 70–96% in these patients [12, 13, 38, 39]. On the other hand, patients with ligamentous laxity have less anatomic restraint to prevent dislocation and, as a result, have a higher rate of PF instability episodes but tend to have less severe articular cartilage damage [42]. Coexisting pathologies of trochlear dysplasia, patella alta, excessive tibial tubercle–trochlear groove (TT–TG) distance, and patholaxity of the medial patellofemoral ligament (MPFL) with recurrent patellar instability are frequently observed. Although there is evidence to suggest a strong correlation between the number of patellar dislocations and the prevalence of PF OA [17, 39], it is important to note that a history of patellar dislocation is notable in 33% of isolated PF OA patients [22].

Anatomic abnormalities including trochlear dysplasia, patella alta, and excessive TT-TG distance can be found in isolated PF OA patients without a history of patellar dislocation. In the setting of patella alta, excessive load of the distal patella can occur due to decreased engagement of the patella in the trochlea. The concentration of load on a smaller area of the cartilage results in an increase in pressure and risk of cartilage wear. Rotational malalignment in the axial plane resulting in excessive TT-TG and patellar tilt increases lateral patella facet pressures, predisposing the lateral PF joint cartilage damage. Increased TT-TG values have been shown to be proportional to the development of PF cartilage damage and OA [47]. Finally, high-grade trochlear dysplasia is present in the large majority of patients with isolated PF OA [22]. The presence of the trochlear prominences as observed in Dejour types B–D can lead to high contact loads within the PF joint during the early degrees of flexion [50].

Nonoperative Treatment

Nonoperative treatment for PF OA is focused on restoring strength, balance, and flexibility. A comprehensive rehabilitation program should first aim at restoring range of motion and flexibility, followed by gait training and a strengthening program that targets the core, pelvis, and quadriceps. Other nonoperative treatment options include nonsteroidal anti-inflammatory medications, intraarticular corticosteroid, and viscosupplementation injections, taping, and bracing. Although currently available studies on intraarticular platelet-rich plasma injections for knee OA have reported improvements in pain and knee function, the prevalence of PF OA predicts less reliable improvement in symptoms [24]. Commercial harvesting of stem cells from the bone marrow or adipose and delivery as an injection is available as another nonoperative treatment, although the data on this is currently limited. The use of McConnell taping and Kinesio taping has demonstrated equivocal results [28], but if patients experience symptomatic relief, then it may aid in allowing them to participate in their physical therapy and strengthening program.

Operative Treatment

Tibial Tubercle Osteotomy (TTO)

Tibial tubercle osteotomy (TTO) is a highly versatile operation that can be used to correct coronal plane malalignment in the setting of instability and elevated tibial tubercle to trochlear groove (TT-TG) distances, as well as to unload painful or damaged cartilage. It is often used in conjunction with cartilage restoration procedures of the patella or trochlea to unload the chondral repair site. Straight anteriorization

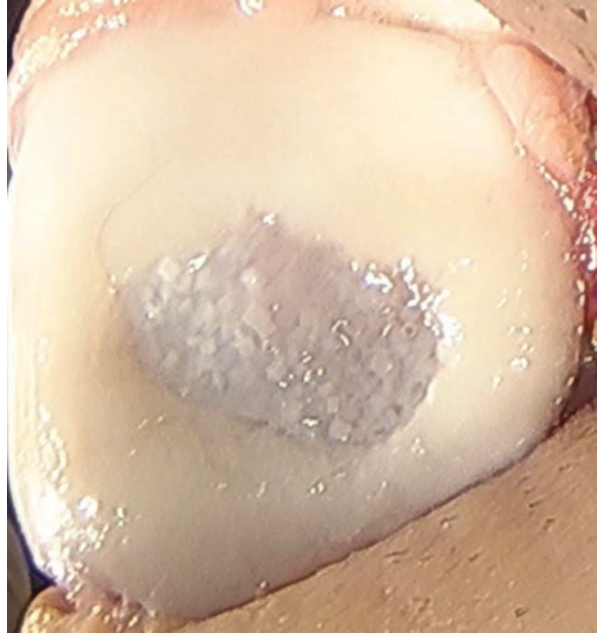
of the tibial tubercle (Maquet procedure) by 1.3 cm can decrease inferior patellar loads by approximately 57–84% at all flexion angles [15]. Similarly, straight anteriorization of the tibial tubercle can decrease trochlear loads by approximately 20–32% at all flexion angles [43]. Anteromedialization of the tibial tubercle shifts the contact forces to the medial patella and trochlea and can significantly decrease both peak lateral and total loads across both the patella and trochlea at all flexion angles [3]. However, in the setting of physiological tracking, anteromedialization of the tibial tubercle can potentially increase the trochlea loads in patients with central or medial chondral defects. For autologous chondrocyte implantation (ACI), studies comparing results with and without TTO have confirmed that an unloading osteotomy significantly improves patient outcomes compared with ACI alone [40, 49]. Ultimately, in order to unload the cartilage repair site, the magnitude and direction of translation of the tuberosity at the time of TTO will vary depending on the patient's anatomy, etiology, and location of the chondral defect. Furthermore, in the setting of lateral PF instability, a MPFL reconstruction is recommended in conjunction with the TTO to restore patellar stability [32].

For patients with symptomatic isolated PF OA who have failed conservative treatment, treatment with TTO can improve outcomes. The outcomes of anteromedialization TTO for PF OA are correlated with the location, rather than the severity, of cartilage wear; patients with distal or lateral facet patellar lesions tend to have the best outcomes, whereas those with proximal or diffuse patellar lesions and central trochlear lesions tend to have the worst outcomes [41]. Central trochlear lesions often present with a corresponding patellar lesion, and in these patients, patellofemoral arthroplasty (PFA) may be a better option than combined TTO and cartilage restoration. Atkinson et al. examined 40 patients (50 knees) with a mean age of 29 years who underwent TTO for PF OA and reported that 94% had improved pain scores and 77% had good or excellent results at a mean follow-up of 81 months [2]. Similarly, Carofino and Fulkerson examined 17 active patients (22 knees) with a mean age of 55 years who underwent an anteromedialization TTO for PF OA and reported that 63% had good to excellent results at a mean follow-up of 77 months [8]. Liu et al. examined 57 patients (61 knees) with mean age of 30 years who underwent an anteromedialization TTO for PF OA and reported a return to sport rate of 83% at an average of 8 months postoperatively [27]. Further investigation is needed to determine the long-term complication profile and survival of primary TTO for PF OA.

Cartilage Restoration Procedures

Many of the cartilage restoration procedures available for the treatment of cartilage defects of the PF joint have been adopted from techniques successfully used for femoral condylar lesions, although the clinical outcomes of these procedures for PF

Fig. 4.2 Cartilage restoration with particulated juvenile articular cartilage for a well-shouldered lesion of the patella, which has intact cartilage margins to contain the graft



chondral lesions are often less satisfactory. Clinical outcomes are better in patients who have isolated trochlear defects than in those who have patellar defects [7, 16, 20, 45]. Cell-based techniques have become popular for treating defects of the PF joint due to the ability to contour the graft to match the surrounding articular topography. However, these techniques rely on the underlying subchondral bone to provide a stable base for the graft, as well as intact cartilage margins to contain the graft and prevent it from displacing (Fig. 4.2). Therefore, in the setting of underlying bony deformity, cystic lesions, and loss or uncontained lesions, the use of osteochondral grafts may be a more appropriate treatment option.

Microfracture

Historically, microfracture was viewed as the first-line treatment for chondral defects in the knee. For the PF joint, specialized awls were utilized to allow for perpendicular penetration of the subchondral plate of the patella and trochlea. However, multiple studies have demonstrated deterioration of the initial clinical improvement between 18 and 36 months after the procedure [25, 35]. Although microfracture provides an attractive option due to its ease of use, low cost, and ability to be performed arthroscopically, the questionable durability of the fibrocartilage

tissue produced (especially in the patellofemoral joint, which sees increased shear forces) and its inability to treat larger lesions (>2 cm) have limited the use of the procedure.

Autologous Chondrocyte Implantation (ACI) and Matrix-Induced Autologous Chondrocyte Implantation (MACI)

ACI is a two-stage procedure that first involves harvesting a small amount of cartilage from the non-weight bearing region of the knee, which is then digested and the chondrocytes expanded in cell culture for approximately 3 weeks. During the second stage, the chondral defect is prepared, and a patch (previously the periosteum but now more commonly the collagen) is sewn to seal the defect. The expanded chondrocytes are then injected underneath the patch. For PF chondral defects, the advantages of ACI include the ability to treat large lesions and its ease of use in the PF joint, where the complex geometry can make contouring of other grafts difficult. Although the early results of first-generation ACI for patellar lesions were poor [5], functional outcomes and patient satisfaction were significantly improved when ACI was performed in conjunction with a TTO to offload the PF joint [40, 49]. More recent studies have demonstrated good long-term outcomes of ACI in patients with PF defects, with satisfaction rates of >80% to 90% [18, 19]. However, concerns have been raised about the implantation of chondrocytes in suspension, which may result in the uneven distribution of chondrocytes within the defect, potential for cell leakage, and loss of the normal chondrocyte phenotype. In order to overcome these concerns, matrix-induced autologous chondrocyte implantation (MACI) was introduced and seeds the patients' chondrocytes on three-dimensional porcine type I/type III collagen bilayer scaffold (Fig. 4.3). The early results of MACI in the PF joint are promising [11, 34], although long-term studies are needed to evaluate for any superiority over ACI.

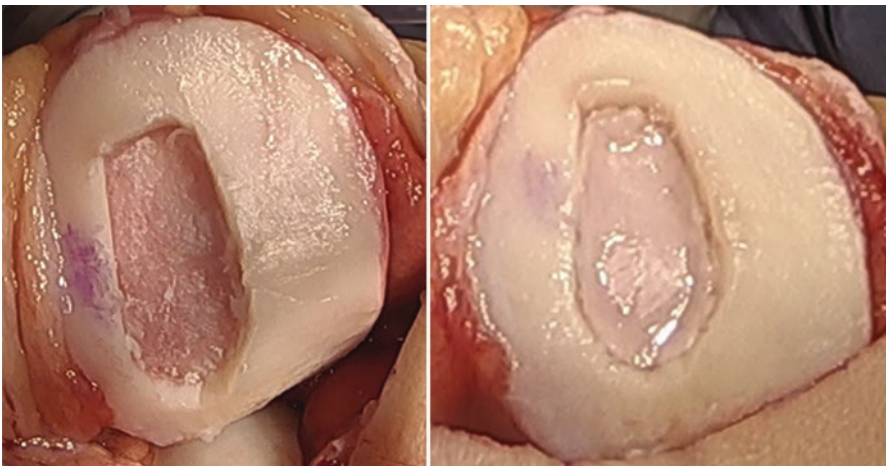


Fig. 4.3 A well-shouldered patellar chondral defect (left) treated with matrix-induced autologous chondrocyte implantation (right)

Particulated Juvenile Articular Cartilage

Another cell-based restoration procedure is particulated juvenile articular cartilage. This technique employs an off-the-shelf source of chondrocytes to resurface cartilage defects, thus providing a single-stage procedure that avoids the morbidity of cartilage harvest associated with ACI and MACI. Cartilage is obtained from deceased juvenile (neonates to 13 years of age) donors, screened, and processed. During implantation, the particulated juvenile articular cartilage is mixed with fibrin glue and is placed in the prepared lesion site (Fig. 4.4). Like ACI and MACI, the particulated graft/fibrin glue construct can be easily contoured to match the articular topography of PF joint. Although cartilage restoration with particulated juvenile articular cartilage for unshouldered patellar lesions has a higher risk of graft displacement in the setting of malalignment-related OA, this technique combined with an offloading TTO can still result in good cartilage fill (Fig. 4.5). Currently, there is limited data on the use of particulated juvenile articular cartilage for the PF joint.

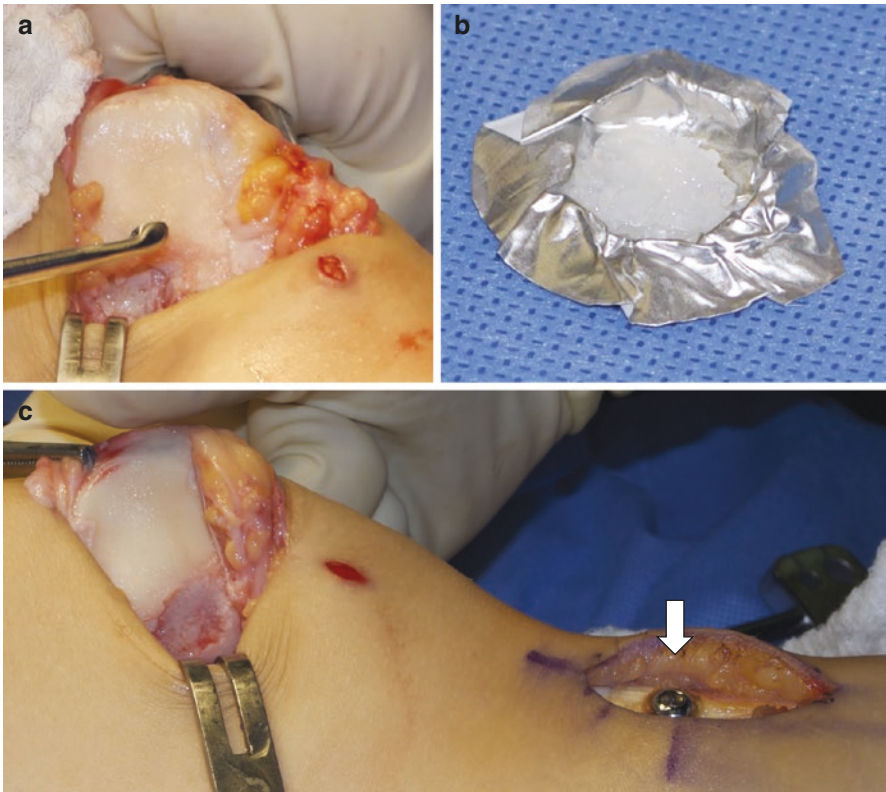
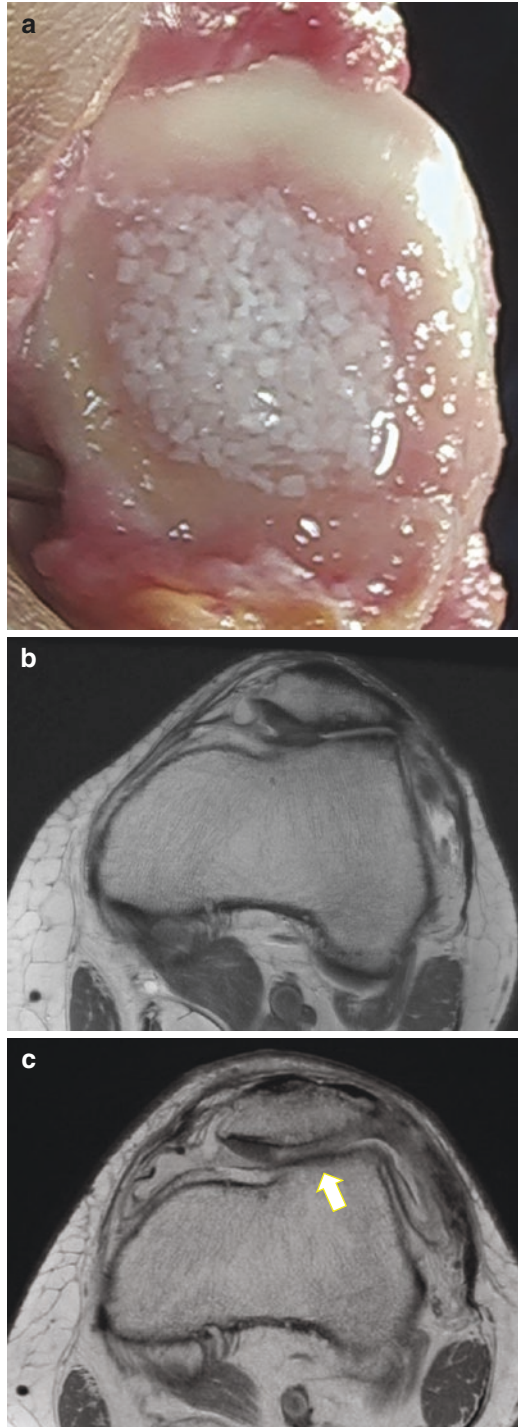


Fig. 4.4 Cartilage restoration of a patellar chondral defect using particulated juvenile articular cartilage. (a) Preparation of the chondral defect with a curette, leaving an intact border of healthy cartilage with stable margins. (b) Creation of the fibrin glue and particulated juvenile articular cartilage mixture in its foil package. (c) Implantation of the particulated juvenile articular cartilage into the patellar chondral defect. A concomitant anteromedialization tibial tubercle osteotomy (arrow) was performed prior to implantation

Fig. 4.5 (a) Cartilage restoration with particulated juvenile articular cartilage for an unshouldered lesion of the patella. In the setting of malalignment-related OA, this generally results in a higher risk of graft displacement. However, in this patient, particulated juvenile articular cartilage was performed in conjunction with an offloading tibial tubercle osteotomy, and (b) preoperative and (c) 3-month postoperative axial magnetic resonance imaging demonstrate interval fill of the patellar cartilage defect (arrow)



Two of the first studies on this procedure, which were funded by the industry developer for DeNovo (Zimmer), evaluated patients treated with particulated juvenile articular cartilage for defects of the patella and reported substantial improvements in clinical outcome scores, reduction in pain levels, and >90% fill of the cartilage defects [6, 48]. Other nonindustry-funded short-term studies have since confirmed MRI evidence of progressive graft maturation over time [21] and histologic evidence of type II collagen production in the repair tissue [14].

Osteochondral Grafts

For lesions with underlying osseous abnormalities (cystic changes) or bone loss, treatment with osteochondral grafts is advantageous due to the single-stage implantation of viable, mature, and structurally stable grafts that replace both the cartilage and the underlying abnormal bone. The biggest challenge with implantation of osteochondral grafts in the PF joint is matching the curvature of the surrounding articular surface of the patella and/or trochlea. Any mismatch in contour between graft and recipient can lead to increased contact pressures if the plug is left proud or rim loading if the plug is recessed. Additionally, the cartilage of the patella is substantially thicker, and the curvature of both patella and trochlea is unique and different than other sites in the knee. Therefore, implanted osteochondral plugs harvested from sites other than the patella can result in the cartilage portion of the graft being thin and the bony portion of the plug extending above the native surrounding subchondral plate, which may create a stress riser and lead to cyst formation and graft failure [51]. For most locations on the trochlea, the curvatures are substantially different from the convex nature of the femoral condyles, and osteochondral grafts that are harvested from the condyles will likely not reproduce the native architecture of the trochlea.

The use of osteochondral autograft transfer (OAT) for PF cartilage defects is limited for patients who have small (<2 cm²) lesions of the patella or trochlea due to the autogenous harvesting of donor plugs. Clinical outcomes have been inconsistent and can be partially attributed to the fact that the autograft plugs can only be harvested from the periphery of the trochlea or intercondylar notch. A few studies have demonstrated significant clinical improvement and MRI evidence of good cartilage fill, complete trabecular incorporation, and fibrocartilage filling of the donor sites in patients treated with OAT of the patella and trochlea [23, 37]. In contrast, within a randomized controlled trial of 100 patients with osteochondral defects of the knee treated with either OAT or ACI, for patellar lesions, 60% of patients treated with OAT ($n = 5$) had an excellent or good result, compared to 85% of patients treated with ACI ($n = 20$). All five patients treated with patellar OAT had failed at final follow-up [4].

Osteochondral allograft transplantation (OCA) does not suffer from donor site morbidity, and size-matched allografts can be requested to optimize the ability to match the topography of the recipient PF joint. Because the limitations associated with donor site morbidity are avoided, OCA can be used to treat large lesions and is frequently used as a salvage procedure after failed cartilage repair. Graft availability (depending on size matching, donor age, and disease screening) and the narrow win-

dow of time in which the graft can be implanted remain the biggest disadvantages of this technique. Fresh osteochondral allografts have demonstrated superiority over frozen allografts, largely because chondrocyte viability has been reported to be critically important for maintaining the biochemical and biomechanical properties of OCA [10]. Chondrocyte viability steadily declines after procurement and falls below acceptable levels (<70% viable cells) by 28 days [52]. Mandatory disease screening requires approximately 14 days, resulting in a narrow window of time (approximately 14 days) for scheduling surgery and transporting tissues. For this procedure, lesions are sized and reamed to a bed of normal bone, and a corresponding dowel is taken from the allograft, contoured to match the recipient site, and gently implanted into place for press-fit fixation (Fig. 4.6). In some instances where press-fit fixation is not attainable, supplemental fixation with the use of headless compression screws or absorbable pins may be needed. Like the results of other cartilage restoration tech-

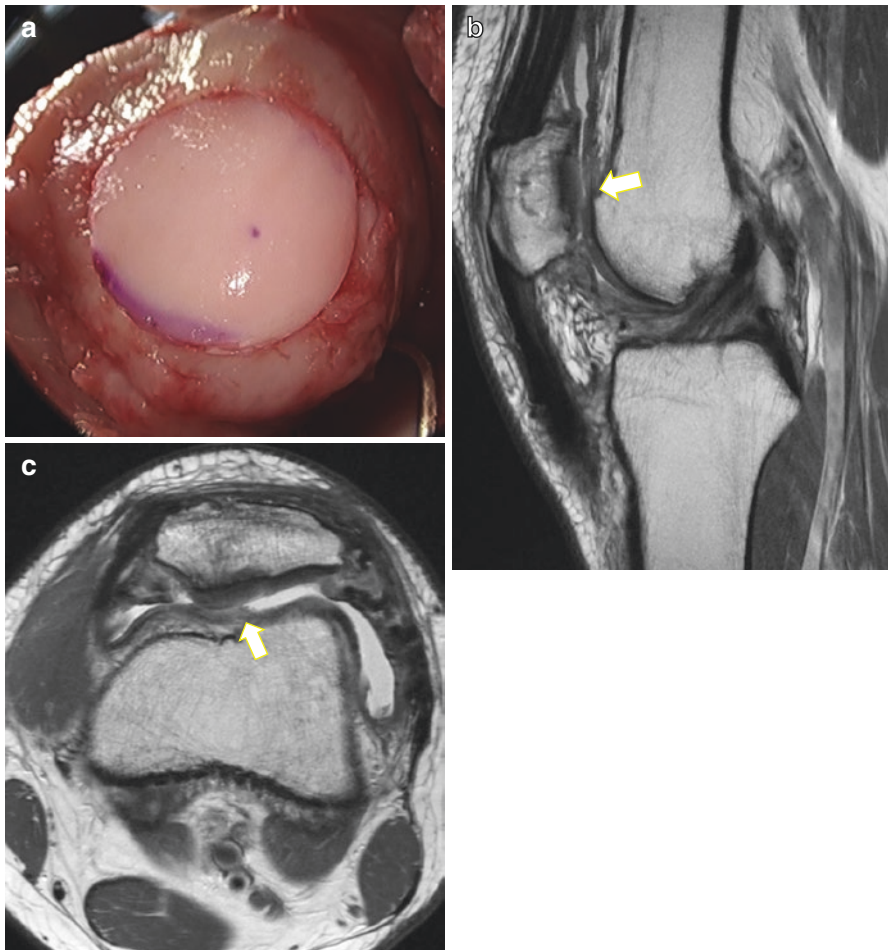


Fig. 4.6 (a) Cartilage restoration of a patellar chondral defect using osteochondral allograft transplantation. (b) Lateral and (c) axial magnetic resonance imaging demonstrates reconstitution of the patellar articular surface (arrow) and partial trabecular integration

niques, graft survivorship and clinical outcomes of OCA in the PF joint are generally inferior to that of the femoral condyle [9]. Bugbee and colleagues reported that in their series of patients, 10-year graft survivorship was 78% for isolated patellar defects and 92% for isolated trochlear defects, with significant improvement in clinical outcome scores in both groups of patients [7, 20]. For bipolar lesions treated with OCA, high reoperation and failure rates have been reported [33].

Patellofemoral Arthroplasty (PFA)

Since its introduction in 1955, patellofemoral arthroplasty (PFA) (Fig. 4.7) has evolved in sophistication and efficacy. PFA performed with the use of first-generation implants resulted in revision rates as high as 63% [46]. Over time, improved prosthetic design and patient selection have led to improved patient outcomes [29,

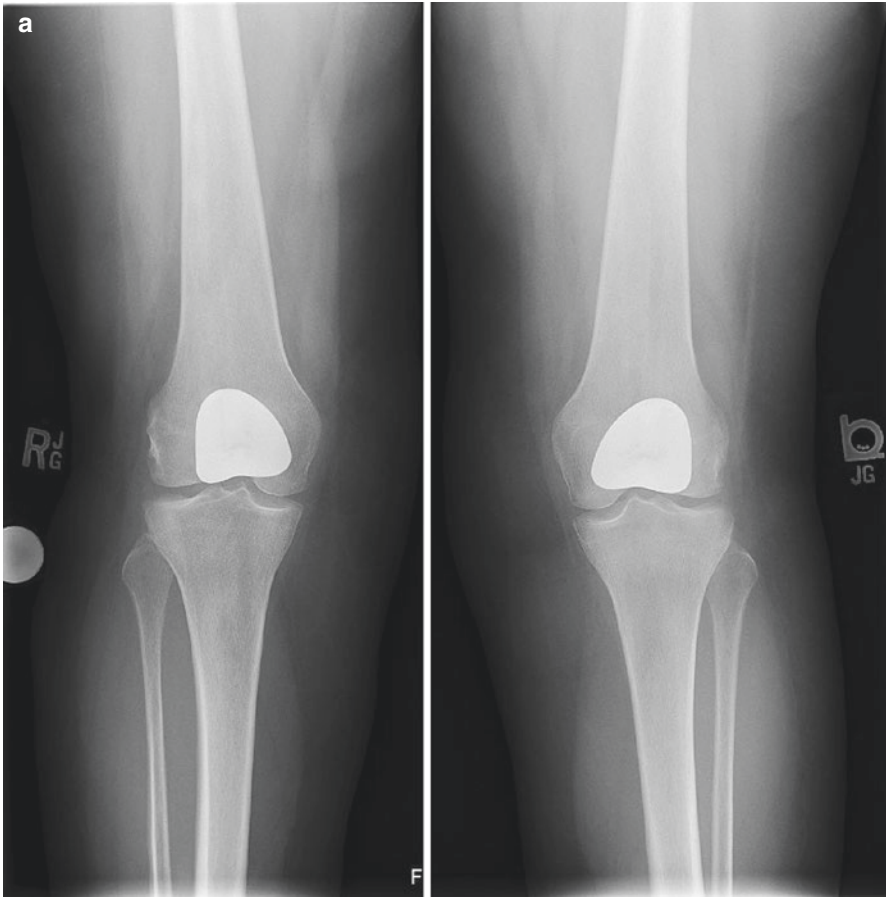


Fig. 4.7 (a) Anteroposterior, (b) lateral, and (c) merchant postoperative standing X-rays showing a successful patellofemoral arthroplasty

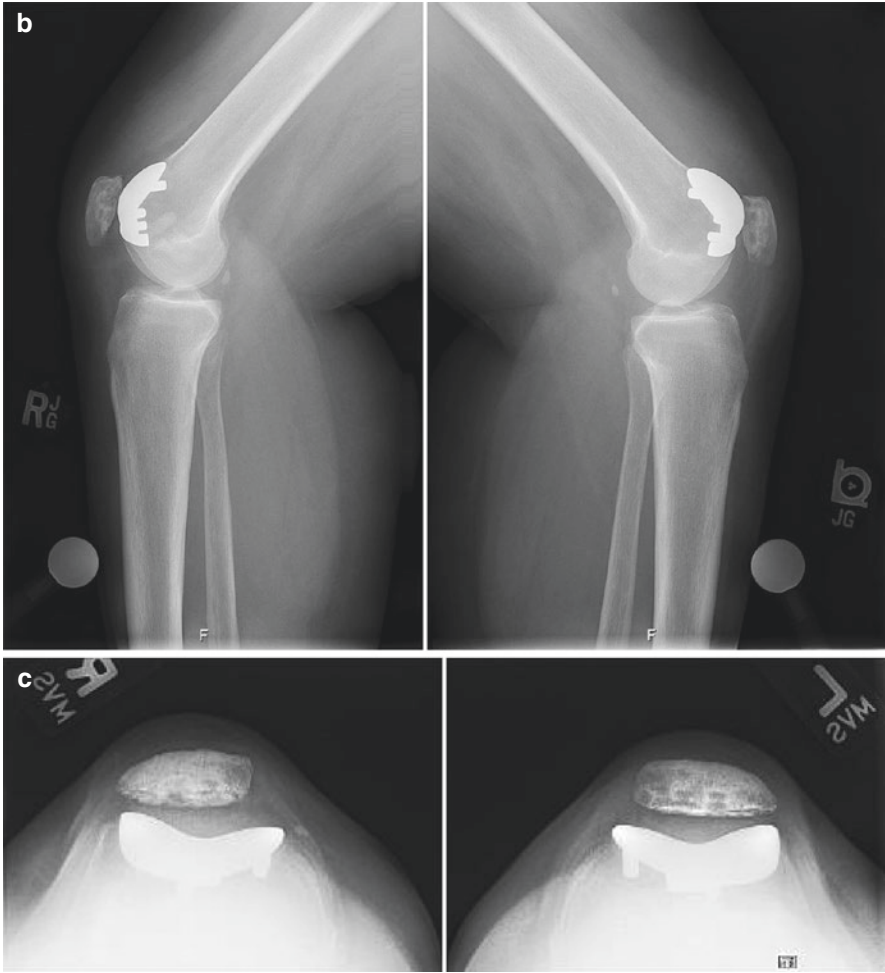


Fig. 4.7 (continued)

31]. With any PFA, there is always the risk for progressive tibiofemoral arthritis in the remainder of the knee. However, unlike its medial and lateral unicompartamental counterparts, survivorship of PFA is closely tied to the etiology of the disease, with posttraumatic, malalignment, and instability-related degenerative joint disease faring significantly better than primary osteoarthritis. The reason for the improved survivorship seen in patients with posttraumatic, malalignment, and instability-related OA is that the source of the arthritis is clear and limited to the PF joint, and therefore, the risk of progression to the uninvolved tibiofemoral compartments is less likely. In patients with primary OA of the PF joint without trochlear dysplasia

or one of the above etiologies, the OA is presumed to be the initial presentation and so will commonly go on to affect the tibiofemoral joint at some point in the future. PFA is still a good option in young patients with primary OA who do not presently exhibit tibiofemoral OA on MRI; however, they should be counselled that a PFA is likely a bridging operation that will at some point require conversion to a total knee arthroplasty (TKA). Although TKA may be an effective treatment option [26, 36], PFA has many advantages over TKA for the treatment of isolated patellofemoral arthritis. It is less invasive, requires shorter tourniquet times, has less blood loss, has a faster recovery, preserves native knee kinematics, and is bone conserving [29].

Compared to first-generation PFAs, newer designs have features that optimize patellar tracking which has solved some of the earlier problems of patella catching and recurrent instability. Most notably, there is a longer proximal trochlear component and wider anterior flanges, which prevents the patella from jumping onto the trochlear component from the native femur during early knee flexion. Currently, there are two styles of PFA: inlay and onlay. The inlay-style component is set into the anterior trochlear surface, while the onlay prosthesis is implanted flush to the anterior femoral cortex (thus requiring an anterior femoral cut similar to that of a total knee arthroplasty). Though the inlay design resects less bone, it does not allow for any change in rotation of the trochlear component relative to the patients' own anatomy (which often times is pathologic in this group). Thus the inlay style has a higher tendency for patellar maltracking [30]. Both onlay and inlay components allow for creation of a trochlear groove when the native femoral trochlea is dysplastic. However, in patients with a previous or current history of patellar instability, the onlay design is preferred given the ability to increase external rotation of the trochlear implant.

Severe coronal deformity, if left uncorrected, can negatively affect patellar tracking and predispose to progression of tibiofemoral arthritis after PFA. In addition, a PFA cannot completely correct a severely malaligned or unstable patellofemoral joint. Therefore, candidates for a PFA who have an elevated TT-TG distance combined with a history of previous or present instability should be considered for a concomitant MPFL reconstruction. We prefer to use an onlay-style PFA as it allows the surgeon to change the rotation of the femoral component to some degree, which can be very helpful in cases of PF OA due to instability. Furthermore, it is important to recognize patella maltrackers early and perform PFA before significant erosion (Fig. 4.8a) and patella acetabularization (Fig. 4.8b) occur, which can result in insufficient patellar bone for implanting a patellar component. In patients with maltracking-related PF OA and fixed tilt, we use a lateral parapatellar arthrotomy combined with a lateral lengthening which allows us to address the tilt concomitantly. The lateral approach with lengthening can be done in patients with isolated maltracking OA and in those with malalignment and instability. In those cases with combined instability, we add an MPFL reconstruction in addition to the lateral lengthening (Fig. 4.9).

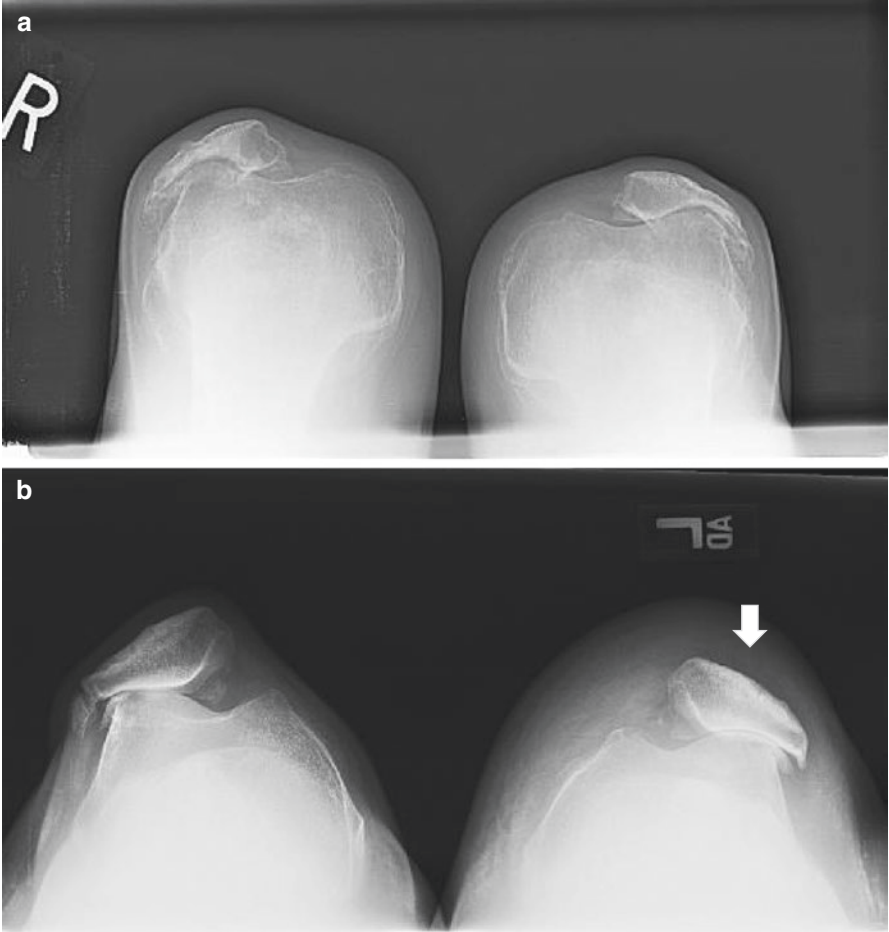


Fig. 4.8 A merchant view showing (a) severe patellar bone loss bilaterally and (b) patella acetabularization of the left knee (arrow)

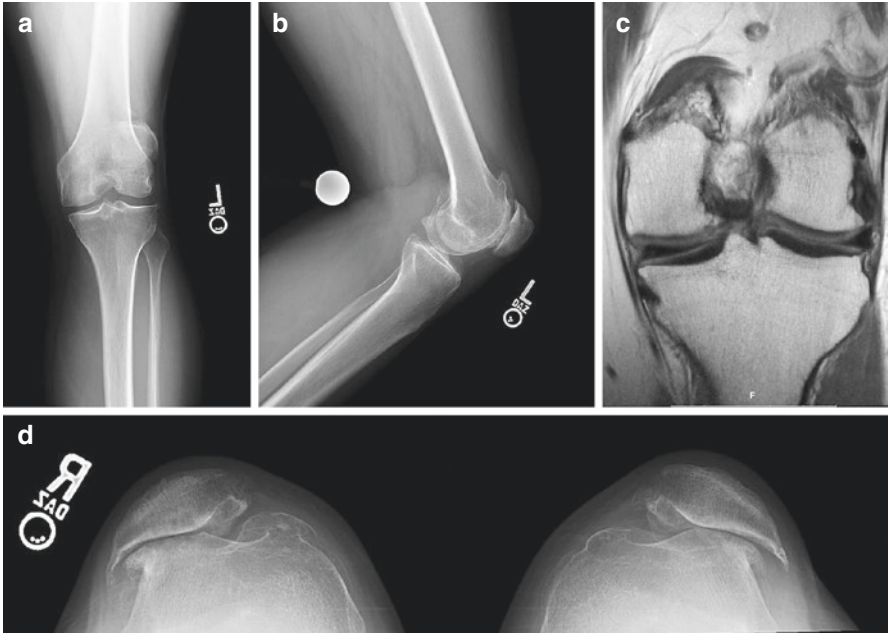


Fig. 4.9 (a, b, d) Preoperative posteroanterior, lateral, and merchant views of the left knee of a patient with maltracking-related patellofemoral osteoarthritis and fixed patellar tilt, (c) coronal magnetic resonance image demonstrates preserved tibiofemoral cartilage and joint space, (e-g) postoperative posteroanterior, lateral, and merchant views of the left knee in the same patient after combined patellofemoral arthroplasty through a lateral parapatellar arthrotomy, lateral lengthening, and medial patellofemoral ligament reconstruction

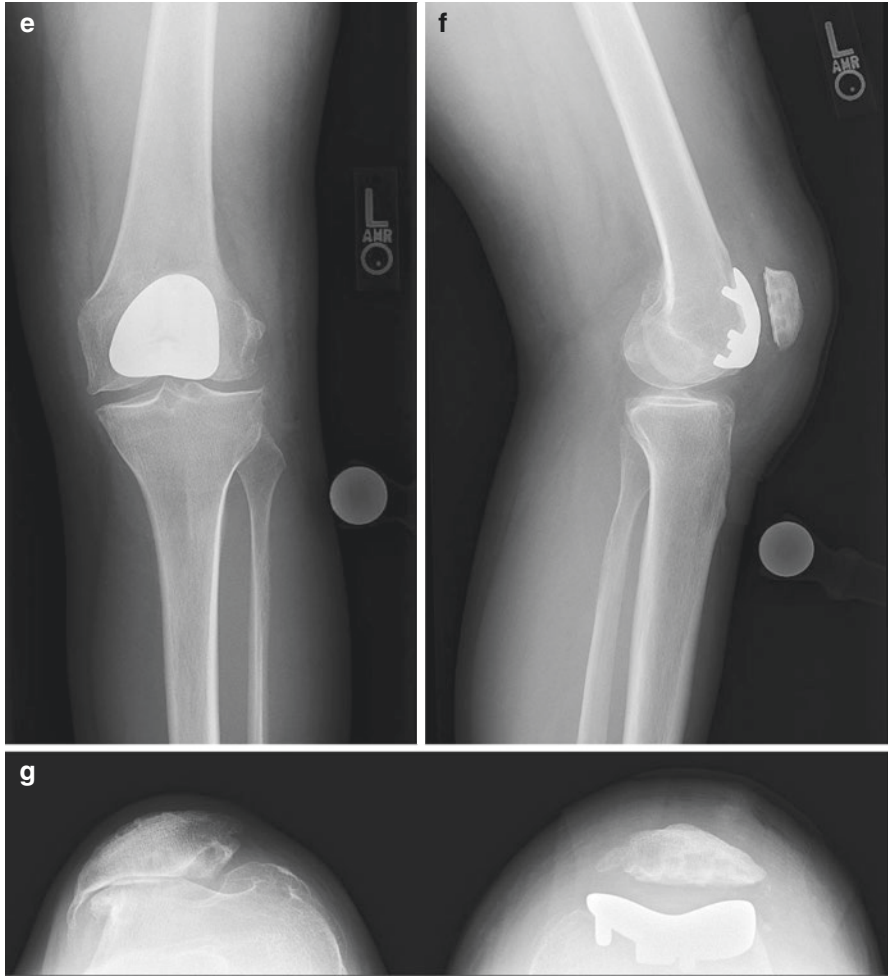


Fig. 4.9 (continued)

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