42 Cartilage Restoration in the Patellofemoral Joint

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42.1 Introduction

The appropriate treatment of patients with anterior knee pain starts with a thorough clinical history and a carefully conducted physical examination. A thorough assessment of the chondral defects and concomitant patho-mechanical factors is critical to the success of any restorative procedure. Co-morbidities such as patella alta, trochlear dysplasia, increased lateral position of the tibial tubercle relative to the femoral sulcus, and secondary soft tissue problems, such as a hypoplastic vastus medialis muscle or a contracted lateral retinaculum must be clearly defined. The standard treatment algorithms used for tibial or femoral chondral lesions cannot be directly extrapolated to the patellofemoral articulation. As an example, Brittberg and Peterson et al. reported successful outcomes for the tibiofemoral joint with autologous cultured chondrocyte transplantation (ACT); however, the same technique reported suboptimal outcomes in the patellofemoral joint as concomitant pathology such as malalignment was not addressed.[1] Peterson et al. next added treatment of the comorbidities to PF ACT and reported markedly improved outcomes.[7]

[AU2] Chondral defects in the patellofemoral joint have varied etiologies. For example, the chondrosis may be genetically related, as with focal or diffuse degeneration secondary to trauma (direct impact or a result of patellofemoral instability) or secondary to repetitive microtrauma (e.g., excessive loads such as in jumping sports), or related to the cumulative microtrauma of biomechanical abnormalities (e.g., chronic patellar subluxation). High-grade (grades III and IV) focal chondral defects (Table 42.1) are reported to occur between 11% and 20% in patients undergoing knee arthroscopy. Of these defects, 11–23% involved the patella and 6–15% were trochlear.[1,10,20] Not all of these lesions were symptomatic. In fact, some patients are asymptomatic even at very high functional levels. Kaplan et al. performed MRIs on asymptomatic NBA basketball players and found articular cartilage lesions in 47% of these players, with 50% of these lesions classified as high grade (III or IV). The patella was affected in 35%, and the trochlea in 25% of these players who were asymptomatic.[23] Similarly, Walczak et al. found abnormal cartilage signal on MRI in 57% of asymptomatic NBA players with a 7% incidence of focal defects.[42]

Just as with other PF problems, symptoms and pathology have incomplete correlations. It is not entirely clear why some patients with PF chondral lesions present with pain while others can perform at a high level. Ficat and Hungeford proposed that the elevated intraosseous pressures seen in the face of an articular cartilage lesion could be the source of pain and today with MRI, it is not uncommon to see areas of bone overload associated with chondral lesions as evidenced by "bone bruises."[31] As noted, the articular cartilage is aneural, so pain other than bone may emanate from the soft tissues, including the joint capsule, ligaments, tendons, and synovium. In addition to mechanical factors, pain may be initiated in part by irritation from chondral debris, which activates an inflammatory and nociceptive response. As the true pain generator is often not well defined,

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Table 42.1 Summary of modified outerbridge and ICRS chondral grading scales

Grade	Modified outerbridge	ICRS
Grade 0	Normal	Normal
Grade 1	Softening	A: Near normal B: Soft intact or superficial open lesion
Grade 2	Open fissures, fibrillation to 50% depth	Abnormal lesion to <50%cartilage depth
Grade 3	Open fissure fibrillation to palpable bone (>50% depth)	Severely abnormal A: >50% cartilage depth B: Down to calcified layer C: To but not through bone
Grade 4	Exposed bone	Severely abnormal full-thickness cartilage loss and bone loss
Notes		Add size and site of lesion

it is crucial to thoroughly evaluate all potential sources of discomfort before attributing symptoms to a chondral defect. This chapter presents the systematic approach to decision-making process for, and surgical treatment of, chondral defects of the PF compartment.

42.2 History

A careful clinical history is the first step necessary to make an accurate diagnosis. Patients may report a history of either an insidious onset of symptoms or acute onset after trauma. In addition, it is not uncommon for the mechanism of the PF chondral pathology to be unknown. In general, patients with patellofemoral pathology can be divided in two main groups: anterior knee pain and patellar instability. It is critical to determine which of the patient's symptoms are most prominent: pain or instability, noting that these are not mutually exclusive. The approach to these two subsets is somewhat different and thus the need to fully explore the patient's main complaint is presented. The character of the anterior knee pain is important to elicit from the patient. Pain from the anterior soft tissues is often described as acute, episodic, and/or localized/poorly localized. Document what activities and positions aggravated the pain. The patient can often perform maneuvers in the office to reproduce pain. Pain due to a chondral etiology mediates through the same tissues that cause pain for other PF pathologies. It is often poorly localized and may be exacerbated by prolonged sitting ("movie theatre sign"). Severe, unrelenting pain that is out of proportion to the patient's exam could be suggestive of a more possible complex regional pain syndrome.

Any history of knee trauma is important. Direct impact injury such as a slip, fall, or "dashboard" type injury may result in anterior knee pain and may damage the patellar or trochlear cartilage even without bone injury. Patellar dislocations may cause damage to the distal medial patellar cartilage and/or lateral femoral condyle. An indirect mechanism is seen with posterior cruciate ligament (PCL) injury. With the posterior displacement of the tibia, PF compartment joint reactive forces are increased.[13] Over time this patellofemoral overload may lead to chondral changes and symptoms of anterior knee pain.

When evaluating the patient with a chief complaint of instability, it is important to determine the amount of energy associated with the first dislocation episode. If the initial episode was a very low energy episode, it should trigger the physician to carefully evaluate for predisposing factors such as generalized ligamentous laxity, patellar alta, trochlear dysplasia, or malalignment. For higher energy dislocations, there is increased risk of chondral pathology with a lower likelihood of significant predisposing anatomic factors. The frequency of and most recent dislocation episode should be recorded, as well as the degree of pain and effusion between instability episodes. Interval pain and effusion symptoms may suggest chondral damage from the recurrent dislocations. In the patient with frequent dislocations, not only is there patholaxity of the soft tissue restraints, there may be significant PF dysplasia and patella alta that will necessitate a more comprehensive reconstructive procedure. It is important to determine whether the patient is having true dislocation episodes or if they are experiencing subluxations or another phenomenon such as "giving way".[13] This may, indeed, represent patellar instability, but can also be related to a pain reflex causing quadriceps inhibition, secondary to ligament deficiency (e.g., ACL deficiency) or intra-articular effusion that inhibits full quadriceps activation.[37] Patients often report crepitus. This has poor correlation with chondral pathology and may be a result of many factors such as: chondrosis, synovial impingement, or scar tissue. Some patients will also complain of mechanical symptoms of locking or catching. In contrast to the symptoms caused by meniscal pathology, mechanical symptoms from the patellofemoral joint usually occur during activity which loads the patellofemoral compartment such as walking down stairs.

Any previous surgical interventions should be documented. It is ideal if the operative reports and the intraoperative arthroscopy images are obtained and reviewed. The date of the procedure is also very important. If it has been a prolonged period since arthroscopic evaluation, a repeat arthroscopy may be warranted to confirm the diagnosis and to define the lesions by location, region, and grade.

42.3 Physical Examination

A focused and detailed physical examination is just as important as the clinical history. The examination entails evaluation of the entire kinetic chain from the foot (pronation vs. cavus) to the tibia (external torsion) to the knee and PF specific exams of the hip. Increased internal rotation increases the suspicion of excessive hip anteversion to the core proximal musculature which will also include the low back and pelvis. Evaluation of muscle weakness of the hip abductors, hip extensors, and pelvic stabilizers is essential. Weakness of these muscles is evaluated by asking the patient to do a single-leg stance on the affected limb which results in a pelvic drop on the contralateral side. Inspection for any deformity or surgical scars should be the first step in the exam. An evaluation of gait should also be performed to assess for any incongruity or abnormality. Both legs should be examined to evaluate for symmetry. The location of the patient's pain should be identified if possible and contributing structures should be carefully assessed. An evaluation for patellar instability should be performed on all patients presenting with symptoms localized to the anterior knee.

Special tests for the PF compartment have been described. Retinacular tightness can be evaluated with the patellar glide and tilt tests. With the knee flexed to 30° the patella is displaced medially. If there is limited medial movement associated with lateral PF facet pain, this is pathognomic of excessive lateral tightness.[13] Lateral retinacular tightness is common in patients with anterior knee pain and is the hallmark of the excessive lateral pressure syndrome described by Ficat.[12] Fulkerson reported tenderness over the lateral retinaculum in 90% of the patients in his series of anterior knee pain.[14] The patellar grind test is an axial compression of the patella on the trochlea and is positive if pain is reproduced and is often positive in the setting of a chondral defect. This test is performed in various angles of flexion in order to establish the location of the chondral defect if present. As the contact area moves from proximal to distal with knee motion, pain near extension is indicative of a chondral defect in the distal part of patella or trochlea; if the pain is elicited at 90º of flexion, the chondral defect is localized to the proximal aspect of patella or trochlea. The sustained knee flexion test is performed by having the patient flex the knee against resistance for 45 s and then having them extend the knee after a period of 15–30 s.[19] The test is considered positive if pain is reported during the extension period. Patellar and quadriceps tendinosis can present as anterior knee pain, so it is important to palpate the proximal and distal poles of the patella. Hoffa's fat pad should be considered as a source of pain especially in the patient who has undergone a previous arthroscopy.[17]

A patient presenting with instability will usually experience a lateral subluxation or dislocation. The patellar glide test is used to assess the medial and lateral displacement of the patella. It is positive if the patella can be significantly displaced in three or more quadrants.[13] Fairbank's patellar apprehension test, when positive, suggests that instability is a significant problem for the patient. The test is positive when the patient has a defensive contracture of quadriceps during lateral patellar displacement at 20º–30º of flexion. Medial instability is often a consequence of an unnecessary or excessive realignment surgery or lateral release. This can be evaluated by Fulkerson's relocation test.[15] This test is performed by holding the patella in a medial direction with the knee extended. The knee is then flexed while the patella is simultaneously released. This causes the patella to relocate into the trochlea. In patients with medial subluxation this test reproduces the patient's symptom. Patellar tracking can be assessed with the "J" sign. The patient extends the knee from 90° of flexion and the patella moves in a proximal and lateral direction that is similar to an inverted "J." This may be an observation in a patient presenting with PF malalignment or in otherwise normal knees. Likewise, a patella that is always lateral may have linear tracking even though there is malalignment.

Tightness of the quadriceps, hamstring, gastrocnemius muscles, and iliotibial band may contribute to anterior knee pain and should be evaluated. Quadriceps tightness is suggested by: (1) a different degree of flexion of one knee (best documented prone) compared to the other, (2) feeling of tightness in the anterior aspect of the thigh, and (3) elevation of the pelvis due to flexion of the hip.[43] Evaluation of iliotibial band tightness is done using Ober's test. To perform this test the patient lies on the nonpainful side and the examiner flexes the affected knee and hip to 90º. The examiner then abducts and extends the affected thigh, which places the iliotibial band on maximal stretch. Palpation of the iliotibial band just proximal to the lateral femoral condyle during maximal stretch will cause severe pain in patients with excessive iliotibial band tightness. To test pelvic tilt, the Thomas test is performed with full hip flexion while observing pelvic movement.

42.4 Imaging

Plain radiographs are a standard part of the diagnostic workup for cartilage-related patellofemoral pain. A standard series includes a standing AP view, 45° (PA "Rosenberg, Shuss or skier view"), a true lateral view, and a low flexion angle axial view (Merchant). The AP view supplemented with a hip to ankle alignment film is used to evaluate the extent of varus or valgus alignment and joint space narrowing. The lateral view is useful for documenting trochlear dysplasia, patellar height (alta or infera), and patellar tilt. Currently, the methods of Caton–Deschamps or Blackburn–Peel are favored over Insall–Salvati. The ratio compares the length of the articular surface of patella and the distance from the most anterior point of articular tibial surface to the most distal point of articular patella surface. Normal ratios are between 0.8 and 1.2. An index greater than 1.3 represents patella alta, and an index less than 0.6 represents patella infera. Dejour et al. have shown that the true lateral radiograph provides more information to assess trochlear dysplasia and patellar tilt than the Merchant view.[2,4,11,17,26] The axial radiograph (Merchant view) is best used to determine information regarding the sulcus angle, joint space narrowing, subchondral sclerosis, and shape of the patella.

CT scan is a useful imaging modality when a tibial tuberosity osteotomy is being considered. The TT–TG (tibial tubercle to trochlear groove) distance can be measured and can guide the surgeon in decision making about the need for an osteotomy. A TT–TG distance of <15 mm is considered normal; values >20 mm are "excessive" and represent malalignment that may be treated with an osteotomy.[4,40] Schutzer et al. identified three patterns of malalignment using CT imaging: type 1 (patellar subluxation without tilt), type 2 (patellar subluxation with tilt), and type 3 (patellar tilt without subluxation).[35] For cartilage specific considerations, a CT arthrogram will allow detailed assessment of lesion position and dimensions. As malposition of the patella relative to the trochlea is often associated with cartilage lesions, additional information may, at times, be obtained from multiple flexion angles (midwaist patellar slices or midwaist patellar cuts comparing quad-active and quad-relaxed views).[34]

Magnetic resonance imaging (MRI) remains essential for the evaluation of osteochondral lesions; in particular the sagittal and coronal image series are useful in evaluating the patellofemoral articulation. MRI has received increased attention due to newly developed high-resolution imaging protocols with the option of enhancement by intravenous gadolinium. This results in sensitivity and specificity approaching 90% for MRI protocols using a 1.5 T magnet with appropriate orthogonal sequences.[32,35,44] MRI also allows assessment of bone overload as evidenced with bone edema (bone bruise).

Bone scans are rarely used in standard cases, but can delineate sites of bone overload or in atypical presentations can be an aid in diagnosing complex regional pain syndrome.

42.5 Basic Science

The lack of vascular, neural, and lymphatic access to articular cartilage creates an environment of limited repair. Injuries that penetrate the subchondral bone initiate a vascular proliferative response resulting in a combination of normal hyaline cartilage (primarily type II collagen) and a structurally inferior fibrocartilage (primarily type I collagen). Each zone of normal hyaline cartilage has a characteristic composition of chondrocytes, collagen, aggrecan, and fluid dynamics that relate directly to that zone's function. Hyaline cartilage consists of 4 zones with the most superficial zone containing the "lamina splendens" (packed collagen fibers) and a cellular layer of flattened chondrocytes. The preservation of this layer is very important for the deeper layers as it limits passage of large molecules between the synovial fluid and cartilage. The intermediate zone is composed of spherical chondrocytes, proteoglycans, and obliquely oriented collagen fibers. The deep zone is a combination of collagen fibers and chondrocytes oriented perpendicular to the articular surface which allows them to optimally resist compressive loads. The deepest level is the calcified cartilage layer which is separated from the deep zone by the tidemark. There are many classification systems used to describe chondral lesions. We have summarized the most commonly used and present them in Table 42.1.

42.6 Patellofemoral Chondrosis Subsets

The cartilage in the patellofemoral articulation is the thickest articular cartilage in the body to accommodate the high loads that are seen in this joint. The variability of bony morphology of this articulation can be a challenge for surgical treatment of the patellofemoral joint. It is useful to categorize patients with PF chondral disease into two categories: those patients with PF chondrosis and associated tibiofemoral chondrosis and those with isolated PF chondrosis. Patients with associated tibiofemoral chondrosis are common. As multiple compartments are affected the outcomes in this patient group are less optimal than in the isolated condition. In order to maximize the outcomes in this patient group the concomitant lesions should be addressed at the same time as the primary procedure. Although, some consideration is given to treating only the most symptomatic lesions as occasionally lesions may be incidental and not clinically relevant. Isolated patellofemoral chondrosis can be divided by etiology into different categories: traumatic, dysplastic, and focal osteochondral defects. The traumatic lesions are subdivided by mechanism into those due to macrotrauma (such as a patellar dislocation or direct blow) and those due to microtrauma (which includes repetitive overuse injuries). Microtraumatic lesions can present with linear fissures of the patella, traumatic delamination, or osteochondral fractures, depending on the degree of knee flexion at the time of the injury. Lesions due to dysplastic conditions result from increased contact pressure. Chondral pathology due to dysplasia can be difficult to treat because of the patellofemoral morphology. These lesions have been treated with soft tissue procedures and TTOs with variable results. Focal osteochondral defects not caused by trauma may be a result of avascular necrosis or osteochondritis dessicans. Lesions secondary to this type of pathology are rare in the patellofemoral joint. Treatment of these lesions requires the surgeon to correct both the underlying bony pathology as well as the chondral defect.

42.7 Treatment and Indications/ Contraindications

42.7.1 Arthroscopic Chondral Debridement

Chondroplasty is indicated in low demand patients who have failed nonoperative treatment with therapy, NSAIDs, and injections or as a staging procedure in patients who may undergo a definitive cartilage restorative procedure. Though not a definitive time period, a trial of nonoperative management for 8–26 weeks is reasonable. This procedure is optimal for those patients with mechanical symptoms without widespread degenerative changes. It may be the first line of treatment in younger patients with chondral defects and can be coupled with procedures such as biopsy for ACI for future interventions or planning a definitive major restoration. Goals of this procedure are to stabilize loose chondral flaps and decrease synovial inflammation from recurrent sloughing of articular debris. Chondral debridement is of questionable value in the truly degenerative knee as several studies have failed to show efficacy over nonoperative treatment.[30]

42.7.2 Microfracture (Marrow Stimulation)

Microfracture is indicated in younger patients with full-thickness, well-contained, small lesions. It is ideally suited for unipolar disease. Though some authors have reported good outcomes with larger PF lesions, other authors report poor results of marrow stimulation for all patellar lesions.[25,38] Most reports suggest treatment for lesions under 4 cm[27,24,29] Perhaps the most important aspect of this surgery is the adherence to the postoperative rehabilitation. This procedure is attractive because it is easy to perform and does not require any additional implants.

Marrow stimulation is contraindicated when there is uncorrected malalignment, global, diffuse degenerative change, or an unwillingness or inability to comply with postoperative rehabilitation demands. A relative contraindication is age >40 years based on the Kreuz outcomes.[25]

42.7.3 Osteochondral Autograft Transplantation

Osteochondral autograft transplantation is indicated for a patellofemoral lesion of less than 2.5 cm^2 when the lesion is contained. It is contraindicated in uncontained lesions, bipolar lesions, or when there is uncorrected malalignment.

42.7.4 Autologous Chondrocyte Implantation/Transplantation

ACI is indicated in symptomatic, full-thickness defects of the patellofemoral articulation and in the United States it is a second-line treatment. Larger lesions and bipolar disease can be treated successfully with this technique. This is a treatment option for larger lesions and those lesions that have failed other techniques (unless extensive bone loss is present) noting that it is unclear which prior procedures may portend a less optimal result.[28] Results appear to be better with unipolar disease, but focal bipolar disease can be successfully treated. The published series of ACI at the PF compartment often includes significant number of concomitant tibial tuberosity osteotomies; however the technique and discussion of this technique are addressed in a separate chapter in this text (Chap. 40). Relative contraindications to ACI exist when there is subchondral bone collapse or bone loss, uncorrectable malalignment, untreated ligamentous instability, advanced age (>55 years), widespread osteoarthritis, and BMI >30.

42.7.5 Osteochondral Allograft Transplantation

Osteochondral allograft transplantation is indicated for patients with symptomatic, large (>3 cm^2), full-thickness osteochondral or full-thickness chondral lesions. It is often used for second-line treatment of unstable and irreparable osteochondritis dessicans lesions, failed osteochondral autograft transfers, failed ACI, or in the setting of subchondral bone collapse. The pathology should be monopolar as bipolar lesion treatment has a much lower success rate. Patients with advanced or diffuse degenerative changes involving one or both of the tibiofemoral compartments are contraindicated for this procedure and are better served by knee arthroplasty.

Authors preference: For pure chondral pathology, ACI is performed concomitantly with straight anterization if the TT–TG is normal or anteromedialization when the TT–TG is excessive. Isolated bipolar disease of the PF joint in very young and severely symptomatic patients who fail ACI may be treated with fresh OC grafting of both the patella and trochlea given the limited alternatives other than PF resurfacing or total knee arthroplasty. Failed ACI of the PF joint can be treated with revision osteochondral allografting.

42.8 Surgical Technique

Prior to any major surgical restoration effort it is important to have a recent and accurate intra-articular evaluation of the patient's anatomy and chondral defect. If recent, high-quality arthroscopic images or video are unavailable, a diagnostic "staging" arthroscopy should be performed at a date prior to the restoration. When ACI is indicated, a biopsy would be harvested at this surgery.

42.8.1 Microfracture

42.8.1.1 Lesion Preparation

Standard arthroscopic evaluation is carried out and the lesion is identified. All other intra-articular pathology should be addressed prior to performing the microfracture. The lesion is debrided with a curette and mechanical shaver to expose the subchondral bone. It is imperative to create a stable "well shouldered" lesion in order to maximize the success of this procedure. A ring curette can be helpful in the patellofemoral joint for this purpose. When preparing a lesion on the undersurface of the patella, it is often helpful to have an assistant provide counter-pressure and stabilize the patella to aid in the preparation of the lesion or perform a miniarthrotomy to allow unencumbered access. It is also important to remove the calcified cartilage layer in order to fully prepare the lesion.

42.8.1.2 Microfracture

[AU5]

Once the lesion is prepared, a microfracture awl is selected that will allow for perpendicular creation of the holes. The goal is to place the holes 3–4 mm apart and to a depth of 2–4 mm or just until fat globules are seen coming from the underlying marrow (Fig. 42.1). It is often helpful to begin the microfracture at the periphery of the lesion and work from outside in to maximize the amount of holes that can be created. Once the holes have been created the arthroscopy fluid pressure is turned down or off to ensure that blood flows from the created microfracture sites. The arthroscope is withdrawn and the wounds are closed in a standard fashion. A recent basic science study suggests the older form of marrow stimulation, drilling, may have theoretical advantages.[9]

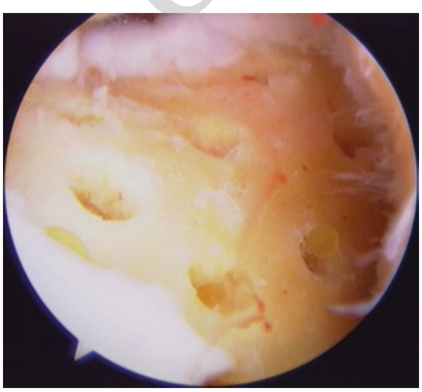

Fig. 42.1 Marrow stimulation of trochlea

42.8.2 Osteochondral Autograft

42.8.2.1 Exposure

The patient is positioned supine on an operating table. The exposure for this procedure is similar to both ACI and osteochondral allografting utilizing a midline utility approach and either a medial or lateral arthrotomy.

42.8.2.2 Recipient Site Preparation

The lesion is sized with commercially available instrumentation and an appropriately sized reamer is selected. The diameter of the reamer should correspond to the diameter of the grafts that are harvested and each recipient hole should be separated by 1–2 mm. Additionally, it is often helpful to allow for an additional 1–2 mm of depth to aid in graft implantation. The holes can be further dilated to facilitate implantation if desired.

42.8.2.3 Graft Harvest

There are three major donor sites available for harvest of autogenous tissue: the lateral femoral condyle above the sulcus terminalis, the superolateral aspect of the intercondylar notch (if uninvolved), or the peripheral aspect of the medial femoral condyle. If the medial condylar donor site is to be used a medial arthrotomy will assist in the harvest. Ideally the largest size plug possible (1 cm^2) is harvested. In the case of larger lesions multiple plugs can be harvested. Most commercially available systems have a T-handle device that is used to gather the donor tissue. This device should be placed perpendicular to the articular surface for the harvest. This device is impacted to roughly 15 mm and the plug is then rotated free from the surrounding tissue. The depth of the graft is measured and used as a guide for creation of the recipient hole.

42.8.2.4 Graft Implantation

The graft remains in the delivery tube and is then placed perpendicular to the recipient site and held in place firmly. It is imperative to limit the number of mallet strikes and the force of each strike when impacting the graft with the plunger as excessive force may lead to chondrocyte death. With the graft nearly completely in place, the tube is removed and gentle impaction is used to seat the graft flush to the surrounding articular cartilage. For larger defects it is beneficial to prepare each graft and recipient site separately until the defect is filled completely.

42.8.3 Autologous Chondrocyte Implantation (ACI)

PF ACI is often performed in conjunction with a tibial tuberosity osteotomy. The technique for AMZ is discussed in detail in other chapters and this section focuses on ACI. In the United States currently matrix autologous chondrocyte implantation (MACI) or other scaffold techniques are not approved for clinical use by the FDA. The current method utilized in the US involves an open procedure with use of a periosteal patch or off label usage of a collagen patch.[8,16]

42.8.3.1 Exposure

A midline utility incision is utilized in all cases. If the patient has a previous anterior knee incision, all attempts are made to incorporate this in the skin incision. As previously discussed this procedure is often performed in conjunction with a tibial tubercle osteotomy and if this is planned, the incision should extend from the proximal pole of the patella to 8 cm distal to the tibial tubercle. If an ACI is performed in isolation, the incision can end at the level of the tibial tubercle. Sharp dissection is carried out through the skin and subcutaneous tissue and full-thickness flaps are created. A lateral arthrotomy allows adequate exposure and may have less morbidity as it spares the vastus medialis. The arthrotomy extends from the level of the vastus lateralis to the anterior capsule, being careful to avoid injury to the anterior lateral meniscus. It is helpful to release the fat pad and to dissect the anterior horn of the meniscus from the capsule as this can increase the exposure of the trochlea. Variable amounts of knee flexion are utilized to maximize the view of the trochlea. The patella is subluxated and or everted medially to expose the trochlea.

42.8.3.2 Recipient Site Preparation

A fresh #15 blade is then used to create vertical walls at the periphery of the chondral lesion. A ring curette is then used to debride the chondral defect and remove all abnormal cartilage, fibrocartilage with preservation of the calcified cartilage layer. It is important to create a "well shouldered" recipient site at this point of the procedure (Fig. 42.2a). Great care should be taken not to gouge the underlying subchondral bone so as to avoid bleeding. Once the lesion is prepared, a template is pressed into the defect and is sized to cover the defect. At this point the tourniquet is deflated and care is taken to achieve homeostasis. Often, especially in the case of revision for a failed microfracture, bleeding is encountered. It is imperative to obtain meticulous hemostasis as bleeding in the recipient site may theoretically lead to reduced production of hyaline-like cartilage. The use of thrombin soaked gel-foam and small neuro paddies can assist in this step. If the bleeding is difficult to control a small amount of fibrin glue can be applied to the base of the recipient site and pressure can be applied for 3–5 min. It is important to ensure that there is adequate surrounding cartilage available to pass suture through in order to provide secure fixation of the patch.

42.8.3.3 Patch Preparation and Fixation

The patch is cut to match the template of the defect. Classically, the patch is sutured by passing interrupted 6-0 Vicryl sutures from the patch through the surrounding cartilage to achieve a water tight seal and the ACI cell suspension is injected deep to the patch (Fig. 42.2b). Suture passage can be facilitated by running the Vicryl through mineral oil prior to sewing.

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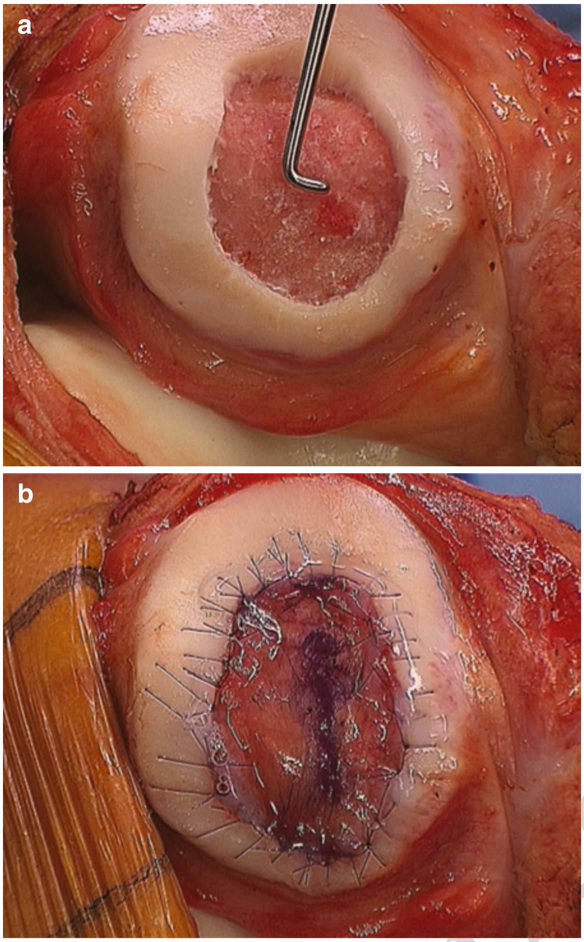

Fig. 42.2 (**a**) The defect is cleared without penetrating the subchondral bone. Walls are vertical. (**b**) The periosteal patch or collagen patch is secured with suture and sealed with fibrin glue. The suspension of cultured chondrocytes is injected deep to the patch

The knots should be tied on the patch side, near the interface of the patch and surrounding cartilage. Occasionally if there is an area that is uncontained, suture anchors can be placed to provide additional fixation. Alternatively, the cells may be seeded on a collagen patch.[39] Ten minutes after seeding, the patch is sewn into place in the same manner as with the classical use of periosteum. A small area may be left open to allow for injection of the additional chondrocyte suspension.

42.8.3.4 Sealing the Patch

Once sutured into place, fibrin glue is used to complete the seal. Care should be taken to use the minimal amount of glue necessary to ensure an adequate seal. If cells are not seeded, the seal can then be tested with saline.

42.8.4 Osteochondral Allografting

42.8.4.1 Preparation of Recipient Site

There are several commercially available systems that can be used to size and prepare both the recipient site and the donor plug. Once the site has been sized a guidepin is placed and a cannulated reamer is used to create a socket with a depth of 6–8 mm. The sidewalls are then trimmed sharply with a fresh #15 blade and the site is irrigated and dried. Once this is complete the depth of the cylindrical socket is measured at the 12, 3, 6, and 9 o'clock positions and this is recorded. Alternatively for a full-area lesion of the patella and/or trochlea, shells may be created free hand.

42.8.4.2 Preparation of the Donor Plug

The ideal donor site is then identified from the fresh osteochondral allograft donor tissue. Though difficult, the goal is to match the native radius of curvature with that of the donor tissue. Once identified the donor is marked with a sizing tube. The donor tissue is fixed in a commercially available jig and a donor-harvesting device is used to core out the donor plug. The graft is then extracted and the measurements made previously of the recipient socket are applied to the donor plug to ensure accurate depth matching.

42.8.4.3 Graft Insertion/Fixation

The recipient site is then dilated an additional 0.5 mm with a commercially available dilation device. The graft is then press-fit into the recipient site with the least amount of force necessary because excessive force has been shown to lead to chondrocyte injury and death. Occasionally an oversized tamp may be required to complete the seating of the graft. For the freehand shell technique, the host bone is cut in the same manner as for a patellofemoral arthroplasty and the shells are shaped to have minimal bone (usually composite thickness of 5–8 mm) with the goal to establish a normal composite thickness (Fig. 42.3a–c). Headless, variable pitch, bioabsorbable screws may be used to fix the shell allografts or to augment the fixation of allograft plugs.

42.9 Postoperative Management

Note that if concomitant tibial tuberosity surgery is performed, the weight-bearing recommendations of that procedure take precedence (see Chap. 40).

42.9.1 Microfracture-Trochlear/Patellar Defect

All patients use continuous passive motion (CPM) from the day of the surgery for a period of 4–6 weeks, 6–8 h per day. Patients with patellar and trochlear groove lesions should be placed immediately in a hinged brace with a 30º–45º flexion stop for at least 8 weeks. Weight bearing in extension as tolerated is allowed immediately postoperatively. After the period of protected flexion, patients begin active range of motion exercises and progress to full flexion. No cutting, twisting, or jumping sports are allowed until at least 6 months after surgery.

42.9.2 Osteochondral Autograft Transplantation

The postoperative management for this procedure is similar to that of both ACI and osteochondral allografting.

42.9.3 Autologous Chondrocyte Implantation of the Patellofemoral Joint

The operative extremity is placed into a hinged knee brace locked in full extension postoperatively. Continuous passive motion is initiated on the first postoperative day (0–30°; 1 cycle/min) in 2-h increments for 6–8 h per day. Range of motion is advanced by 15° each week with the use of the continuous passive motion machine and simultaneous unlocking of the brace. The objective is to obtain 90° of flexion by weeks 6–8, but not generally sooner than 4 weeks.

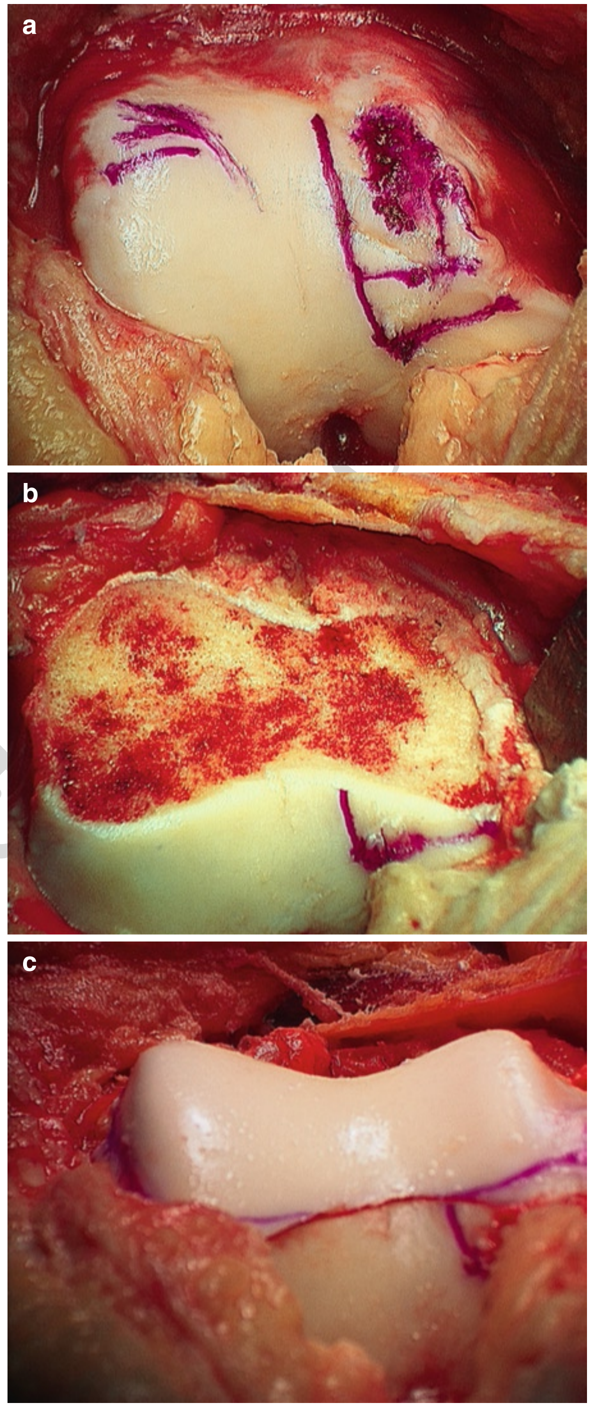

Fig. 42.3 The area of chondrosis is identified (**a**). The bony cuts are made with a cooled oscillating saw (**b**). The donor graft is shaped to fit the defect and secured (**c**)

Return to full activity is not permitted until 8 months postoperatively to protect the lesion until the cartilage has sufficiently matured.

42.9.4 Osteochondral Allograft and Autograft Transplantation

Postoperatively, if the procedure is performed appropriately with a well-contained defect, early weight bearing and motion are encouraged. After a multiple-plug technique, full range of motion and protected weight bearing are advised for the first 4 weeks. At 4 weeks, full weight bearing is allowed. Sporting activities are not recommended until 4–6 months postoperatively.

42.10 Outcomes

A detailed description of the studies evaluating outcomes from the various procedures is included in Table 42.2. Below is a summary of these findings.

42.10.1 Microfracture

Marrow stimulation techniques result in a repair tissue with inferior wear characteristics. Some authors have reported good results in the treatment of smaller defects (2–3 cm^2). However larger lesions and high-demand patients were better treated with autologous chondrocyte implantation or osteochondral grafting.

42.10.2 Autologous Chondrocyte Implantation

Recent results of autologous chondrocyte implantation in the patellofemoral joint have been encouraging with good and excellent results even in patients with large defects (average 10 cm^2) who had previously undergone an average of three surgeries. It appears that concomitant realignment procedures are an important adjunct in obtaining these favorable results.

42.10.3 Osteochondral Grafting: Autograft Transplantation

Transfer of autologous osteochondral plugs is limited by the donor area and donor site morbidity. Based on such limitations, this technique is employed for only small-sized defects. Published studies have reported varying outcomes, suggesting that this technique be suitable for the relatively rare patient who presents with a small isolated chondral defect.

Table 42.2 Summary of various authors' outcomes of specified patellofemoral surgical techniques

Procedure	Authors	Reported outcomes
Microfracture	Blevins et al.[5] Kreuz et al.[25] Steadman et al.[38]	Good results with microfracture in lesions 2–3 cm^2
	Miller et al.[27]	Lysholm score 53.8–83, Tegner score 2.9 → 4.5
	Mithoefer et al.[29] Steadman et al.[38] Kreuz et al.[25]	Good midterm outcomes in approximately 80% of patients
ACI(PF)	Minas et al.[28] Bentley et al.[3] Bitteberg et al.[6]	Good and excellent results in up to 85%
	Yates[45]	No good results without corrective osteotomy 78% good/excellent
OA grafting	Hangody et al.[18] Bentley et al.[3]	Good results 80% of patients Others have shown failure rates that approached 100% when used for patellar defects
	Jakob et al.[21]	86% good/excellent
Allograft transplantation	Jamali et al.[22] Torga et al.[41]	Graft survival in 60–70% of patients with follow-up of up to 10 years
	Shasha et al.[36]	Kaplan–Meier Survival Rate: 5 years – 95%, 10 years – 80%, 15 years – 65%, 20 years – 46%

42.10.4 Osteochondral Grafting: Allograft Transplantation

This type of reconstruction appears to be better suited for the treatment of trochlear defects, though some authors have reported good outcomes with allograft reconstruction of the patella and even bipolar lesions.

42.11 Conclusion

Management of patients with a PF joint that presents with pain and dysfunction associated with chondral pathology remains a difficult clinical problem. Attention to the entire PF system including issues pertaining to alignment is paramount in achieving a successful outcome. Similar to the tibiofemoral joint, all comorbidities must be addressed. Multiple options exist to manage the chondral pathology and are chosen based upon defect size and location. Outcomes support good and excellent results similar to that seen with the management of the tibiofemoral joint.

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