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Focal Chondral Injuries

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Introduction

Focal chondral defects (**FCDs**) constitute a common finding, with a reported incidence of 4.2% and 6.2% in the general population in patients younger than 40, resulting in more than 200,000 surgical procedures per year (Fig. 22.1).[1,2] Furthermore, the prevalence is reported to be as high as 36% in athletes.[3] Importantly, if these lesions are not addressed in a timely manner, they have been reported to worsen over time and may progress to more diffuse osteoarthritis (OA).[4] The treatment of FCD remains a challenge because cartilage repair procedures have failed to reproduce native cartilage to date.[5,6]

The cause of FCD is multifactorial. One well-known cause is osteochondritis dissecans (OCD), a condition in which the subchondral bone and overlying articular cartilage detach from the underlying bony surface, occasionally manifesting as multiple FCD. The incidence of OCD is between 15 and 29 per 100,000.[7] More commonly, FCDs are the result of trauma. Indeed, injuries resulting in acute instability such as knee dislocation and subluxation may also result in the development of articular cartilage lesions. Approximately half of patellofemoral FCDs occur in the setting of a traumatic injury.[8] Additionally, chronic degenerative changes predispose to articular lesions as the result of repetitive microtrauma.

Although cartilage research has grown exponentially, basic science and clinical studies focusing on its foundation, namely the subchondral bone, have not received the same attention.[1] The subchondral bone provides mechanical and biological support for the overlying articular cartilage, and it undergoes constant adaptation in response to changes in the biomechanical environment of the joint.[1] Consequently, subchondral bone lesions are commonly associated with cartilage lesions. An understanding of this anatomy is essential in cases in which the subchondral bone is compromised and recognition of the extent of the lesion may guide treatment and outcomes.

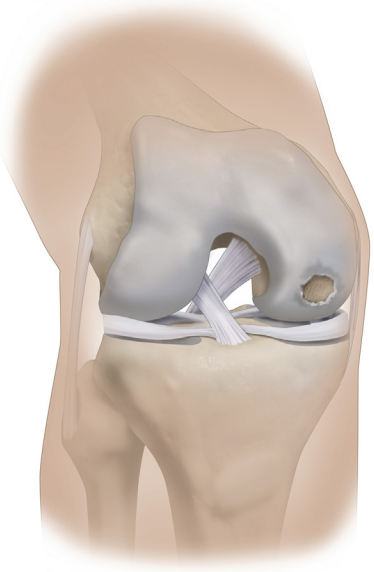
Treatment of FCD has traditionally been managed nonoperatively; however, the literature suggests that somewhere between 11% and 40% of all patients younger than 40 who underwent arthroscopic surgery for other reasons had identifiable and treatable chondral injuries that were unaddressed, which may improve outcomes.[2,9,10] An emphasis on the surgical management of FCD has evolved with the advent of improved biotechnology and surgical techniques to address FCD. In particular, there has been a shift towards cartilage reparative and regenerative procedures in an effort to restore cartilage, prevent further cartilage degeneration, and reduce morbidity.

Several major procedures are considered when treating FCD. These procedures are associated with favourable, reproducible outcomes and include microfracture, osteochondral allograft transplantation (OCA), osteochondral autologous transplantation (OAT), matrix-induced autologous chondrocyte implantation (MACI, Sanofi, Boston, MA, USA), minced cartilage procedures (DeNovo Natural Tissue (NT), Zimmer Inc., Warsaw, IN, USA), viable osteochondral surface allografts (Cartiform, Osiris, Inc., Naples, FL, USA; and Prochondrix, AlloSource, Denver, CO, USA), extracellular matrix scaffolds (BioCartilage, Arthrex, Inc.) and single-stage autologous options (GraftNet, Arthrex, Inc.). Given the array of treatment options, the challenge lies in determining which intervention or combination of interventions is most appropriate given patient- and defect-specific characteristics. As these restorative techniques become more prevalent, it is imperative to provide an update on the outcomes and indications for these procedures to disseminate standards of treatment and to optimize patient outcomes.

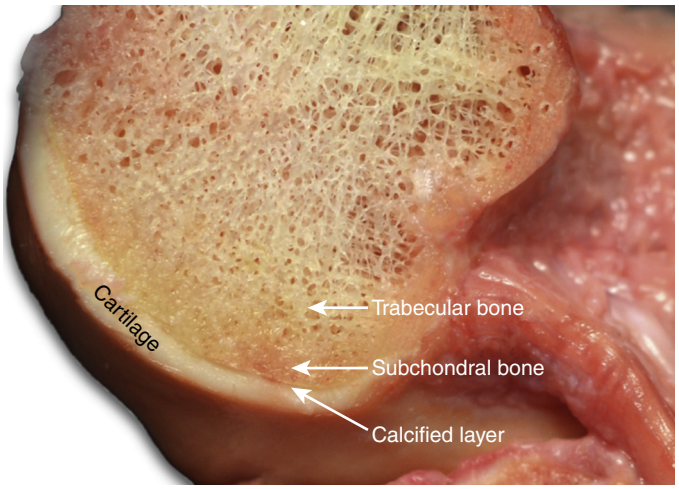
The purpose of this chapter is to provide a comprehensive review of FCDs of the knee and provide the treating surgeon with a thorough understanding of concepts from diagnosis to rehabilitation. In particular, we provide treatment algorithms based on current practice and indications to help guide treatment. Conservative and surgical approaches to the treatment of these defects are described, as well as recommended postoperative rehabilitation. For each surgical approach, a discussion on clinical, radiographic, and outcome survivorship, when available, follow. Finally, future directions for the field of cartilage repair are discussed.

Microscopic Anatomy of Articular Cartilage

Articular cartilage consists of five different zones, which can be distinguished based on the morphology and orientation of collagen fibrils.[2] In the superficial zone (zone 1), the collagen fibres are tangentially oriented into tightly packed parallel laminae that radiate vertically from the calcified zone. Zone 2, or the intermediate zone, contains randomly oriented collagen fibrils. Zone 3, which is also referred to as the radial zone, is the thickest layer with the highest concentration of proteoglycans and water. The tidemark serves as the junction between the calcified and uncalcified cartilage matrix (zone 4). Lastly the zone of calcification (zone 5) serves as an anchor to a complex network of collagen fibrils (Fig. 22.2).



• **Fig. 22.1** Illustration representing a focal chondral defect on the femoral medial epicondyle.



• **Fig. 22.2** Cadaveric dissection image of a hemi-condyle as viewed from the intercondylar notch demonstrating a sagittal view of the superficial and inner layers: cartilage, calcified layer and the differences between subchondral and trabecular bone.

Diagnosis

When undiagnosed or left untreated, FCD has the potential to progress towards further cartilage damage and, according to some studies, OA.[11,12] Once symptomatic, FCDs have a propensity for continued symptom progression over variable periods depending on comorbidities and patient-specific factors. Early management is important to restore normal joint congruity, pressure distribution and normal knee kinematics. A timely diagnosis allows for appropriate consideration of the various treatment options. Because outcomes are highly dependent on the underlying disease, the more precise a diagnosis is preoperatively, the better the algorithm can be tailored to successfully treat these injuries. FCD is diagnosed through a combination of patient history; physical examination; imaging, including plain radiographs and magnetic resonance imaging; biomarker analysis; and arthroscopy.

Patient History

As in any diagnostic work-up, it is important to obtain a comprehensive history from any patient that complains of knee pain, particularly because of the overlapping symptoms of cartilage defects and intraarticular pathological conditions. Sports participation is the most common inciting event shared among those with a diagnosis of chondral lesions.[2] Approximately half of patellofemoral FCDs occur in the setting of a traumatic injury.[8] Patients with FCDs are usually young, active and able to carry out activities of daily living (ADLs), although they may complain of pain with specific activities, such as deep squats or cutting. Notably, an activity or 'trauma' may not necessarily cause the FCD but rather incite the onset of symptoms on a preexisting yet asymptomatic FCD, resulting from localised degeneration.

Pain is the most common presenting complaint for patients with FCDs. This can present acutely in the setting of injury or sudden load or insidiously in the case of repetitive microtrauma or OCD. This is classically reported with weightbearing and localised to the same compartment as the defect.[13] Pain that worsens with flexion suggests a more posterior lesion. Patellofemoral articular defects typically present as anterior knee pain. However, it is not uncommon for patients to report pain located retropatellar, peripatellar or, in the instance of trochlear defects, posteriorly in the popliteal area. Considering that articular cartilage is aneural, the pain often originates from surrounding structures, including capsular or synovial irritation and overload of the subchondral bone resulting in loss of tissue homeostasis.[14] Therefore, if a FCD is identified in the context of pain-free tissue homoeostasis, the structural cartilage defect may not be of clinical significance. If pain and loss of tissue homoeostasis are present, other causes of the pain must also remain on the differential despite a high clinical suspicion.

Activity-related swelling should raise suspicion for a possible FCDs. The presence of this finding in the absence of pain can help exclude other potential pathological conditions. For example, patellofemoral pain syndrome may also present as swelling with activity but is more often than not painful.[13] Activity-related swelling and, in particular, joint effusion indicate more advanced disease.[8] Diffuse cartilage damage more reliably presents with subtle decreases in range of motion, which has a predilection to limit flexion earlier than extension.[15] These patients also present with diffuse rather than focal pain during activity. For the previously mentioned reasons, it is important to differentiate an isolated FCD from an ongoing osteoarthritic process that has patchy diffuse involvement of the cartilage and results in pain as a result of loss of generalised tissue homoeostasis.

To better understand the best course of treatment, it is critical to obtain a history of any previous treatments that the patient has received to the symptomatic knee, particularly previous injections (cortisone, hyaluronic acid, platelet-rich plasma (PRP)) and surgeries. Insufficient rehabilitation or inappropriately timed return to high-load activities after past surgeries is a common source of symptoms when a patient has already undergone an operation on the ipsilateral knee. This should be evaluated as a potential source of pain before considering a costly work-up or revision surgery.

Physical Examination

The physical examination should begin with a gait analysis followed by evaluation of the symptomatic knee joint for effusion, deformity, contracture, malalignment and patellar maltracking. An OCD-derived FCD at the lateral aspect of the medial femoral condyle can cause the patient to ambulate with an antalgic gait or with the affected leg in obligate external rotation (Wilson sign) as a compensatory mechanism to avoid tibial spine impingement.[16] In patellofemoral FCDs, gait abnormalities, such as in-toeing or hip abductor weakness, are commonly seen. Additionally, it is common to see femoral anteversion and valgus malalignment of the lower extremity. Patients with FCDs usually have normal range of motion and focal tenderness over palpable areas along the lateral or medial femoral condyles. Joint line tenderness is commonly elicited when the lesion affects the femoral condyle and tibial plateau. However, it must be recognised that neither a patient's history nor physical examination are sensitive or specific for differentiating cartilage defects from other intraarticular derangements but may only raise clinical suspicion to pursue further work-up.

Imaging

First-line imaging for the approach to FCDs consists of conventional cartilage radiographs – in particular, bilateral standing anteroposterior (AP), 45-degree flexion weightbearing posteroanterior (PA; Rosenberg view), and nonweightbearing lateral and patella sunrise views (Merchant view). These views allow for evaluation of pathological joint conditions, such as degenerative changes in the tibiofemoral and patellofemoral joints, trochlear dysplasia, and abnormal patella height, tilt and subluxation. The Merchant view is useful to determine joint space narrowing in OA of the patellofemoral articulation. In most cases, FCDs will not be apparent on plain radiographs because most lesions are extraosseous; however, this imaging modality can detect lesions that involve subchondral bone and lead to FCD, such as OCD. The 45-degree flexion PA radiographs are particularly helpful to diagnose fairly large OCD lesions along the posterior femoral condyles.[17] When OCD are suspected, contralateral knee radiographs can be considered given the high incidence of bilateral involvement.[18] Radiographic findings consistent with an OCD include an area of osteosclerotic bone, with a high-intensity line between the defect and epiphysis. Radiographs should be assessed for radiolucencies, subchondral cysts, sclerosis, fragmentation, loose bodies, joint space narrowing and physeal status because these can affect the treatment algorithm. The long-leg axial alignment radiograph is the final view implicated in suspected FCD evaluation and is of utmost importance to determine the mechanical alignment in patients with known or suspected chondral defects. The benefit of this view is that it confers the ability to determine whether the symptomatic knee requires malalignment correction with a concomitant osteotomy. It is useful to obtain these images with a radiological marker alongside the knee to allow for correction of magnification and accurate determination of appropriately sized donor tissue if needed depending on surgical approach.

Magnetic resonance imaging (MRI) is an effective imaging modality for evaluating articular cartilage and the subchondral bed. Although determining the size of a lesion on imaging is helpful for prognostic and surgical planning purposes, MRI often underestimates lesion size by as much as 60%.[19] Moreover, the appearance of cartilage lesions on MRI is often inconsistent with clinical symptoms and arthroscopic findings.[20] Novel MRI techniques such as the delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) and T2 relaxation time mapping have shown great potential in the evaluation of articular cartilage, although these technologies are not widely available and not routinely used for clinical purposes. dGEMRIC tags glycosaminoglycan (GAG) content in cartilage and can be helpful in the diagnosis of early knee OA[21] and cartilage health after ligament rupture.[22] This imaging modality also allows for measurement of compressive stiffness after cartilage repair procedures.[23,24]

T2 relaxation time mapping, an emerging MRI parameter that represents the internuclear reaction secondary to transverse relaxation of hydrogen ions,[24] has demonstrated potential in measuring the collagen content of cartilage.[25] The benefit of this test is the ability to evaluate cartilage degeneration from time of injury, thus providing information about optimal timing for surgical intervention. Such advances in imaging allows for the ascertainment of objective information, which helps to determine the optimal window and treatment methodology. However, because they are emerging technologies the authors of this chapter do not routinely use these MRI-based modalities in the evaluation of patients with FCD.

Biomarkers

Cartilage biomarker analysis is a novel diagnostic tool for detecting the presence of chondral damage. Several type II collagen degradation markers, specifically neoepitope specific for type II collagen cleavage (C2C), cartilage oligomeric matrix protein precursor (COMP) and C-propeptide of type II procollagen (CPII), as well as type II collagen synthesis markers (serum PIIANP), have been identified as potential indicators of cartilage degradation.[26] These precursor proteins are processed by proteases before incorporation into fibrils, thus releasing markers that are then detectable in serum and urine assays. The relative levels of collagen synthesis biomarkers and degradation products can be used to gauge the extent of cartilage turnover occurring at the articular surface.[27]

A number of assays exist that are specifically designed to detect these propeptide levels. Although initial studies have reported promising results in the detection of osteoarthritic changes in the joint, there is a paucity of literature that affirms the efficacy of the use of these biomarkers in detection of FCDs. Future studies in this area are underway to validate these biochemical markers as a tool for early detection of morphological changes within the joint with the hopes to help guide the timing and nature of future treatment.[28]

Diagnostic Arthroscopy

Diagnostic arthroscopy remains the gold standard for the evaluation of intraarticular pathological conditions of the knee. This minimally invasive diagnostic procedure allows for direct identification and classification of FCDs, as well as detection of any concomitant injuries or additional articular cartilage pathological condition that may need to be addressed in place of or in addition to the FCD. A simple **debridement** during the procedure may help improve symptomatic lesions and delay treatment of FCDs in almost 60% of patients.[29] During arthroscopy, the chondral defect size can be measured and graded according to standardised criteria systems: the Outerbridge or International Cartilage Repair Society (ICRS) criteria (Table 22.1).

TABLE 22.1 The Outerbridge and ICRS Classification for Joint Cartilage Damage

Grade	Outerbridge Grading System	International Cartilage Rating Systems (ICRS)
0	Normal	Normal
I	Cartilage with softening and swelling	Superficial lesions, fissures, cracks, indentations
II	Partial-thickness defect with fissures on the surface that do not reach subchondral bone or exceed 1.5 cm in diameter	Fraying lesions extending down to <50% of cartilage depth
III	Defect extends to level of subchondral bone with a diameter of more than 1.5 cm	Partial loss of cartilage thickness, cartilage defects extending down >50% of cartilage depth
IV	Exposed subchondral bone head	Complete loss of cartilage thickness, bone only

The size and location of the lesion play a large role in management; therefore it is important to directly document and probe these lesions during arthroscopy. Defect size, patient factors and subchondral bone involvement ultimately contribute to the treatment decision.

Nonsurgical Management

Nonsurgical management of symptomatic FCD of the knee has a limited role given the underlying mechanism of the defect and the biological nature of cartilage, which limits self-resolution. Nonsurgical management of these defects is incapable of restoring the loss of articular cartilage because of the poor intrinsic capacity for healing inherent in cartilage. This is especially true once the FCD is directly correlated as a symptom generator that impairs activity levels and causes sufficient pain or swelling. Alternatively, nonsurgical management, when successful, can lead to transient pain relief in patients with symptomatic focal FCD[30] but is unlikely to provide long-term relief.

Nonsurgical management of FCD of the knee consists of a set of noninvasive options with the intention to maintain function and minimise pain. The use of nonsteroidal anti-inflammatory (NSAID) medications, chondroprotective agents (glucosamine, chondroitin phosphate), intraarticular injections (corticosteroids, hyaluronic acid, PRP), weight loss, physical therapy, activity modification and knee braces may all provide symptomatic benefit in these patients, depending on the severity and progression of the disease. However, it is important to note that these agents do not diminish the rate of progression of cartilage loss, nor do they restore the structural integrity of the articular cartilage.[30]

The long-term results of conservative management are not well studied. In a prospective study of 28 athletes with isolated chondral defects confirmed radiographically, Messner and Maletius[31] investigated the long-term outcomes of these patients to better understand prognosis. At 14-year follow-up, the majority of patients (78.6%) endorsed good or excellent knee function; however, more than 50% of these patients demonstrated interval increases of abnormal findings, with 12 patients demonstrating joint space reduction. The authors concluded that conservative treatment was not useful for modifying disease progression, despite maintaining self-perceived function.

Surgical Treatment Algorithm

An increasing body of evidence suggests that symptomatic FCDs need to be addressed surgically because of the potential for both worsening of associated symptoms and further progression of cartilage degeneration.[13,31,32] Moreover, full-thickness FCDs have been associated with a greater risk of total knee arthroplasty (TKA) compared with moderate OA.[33] Therefore it is imperative for the treating knee surgeon to understand indications and approaches for the surgical intervention of FCD.

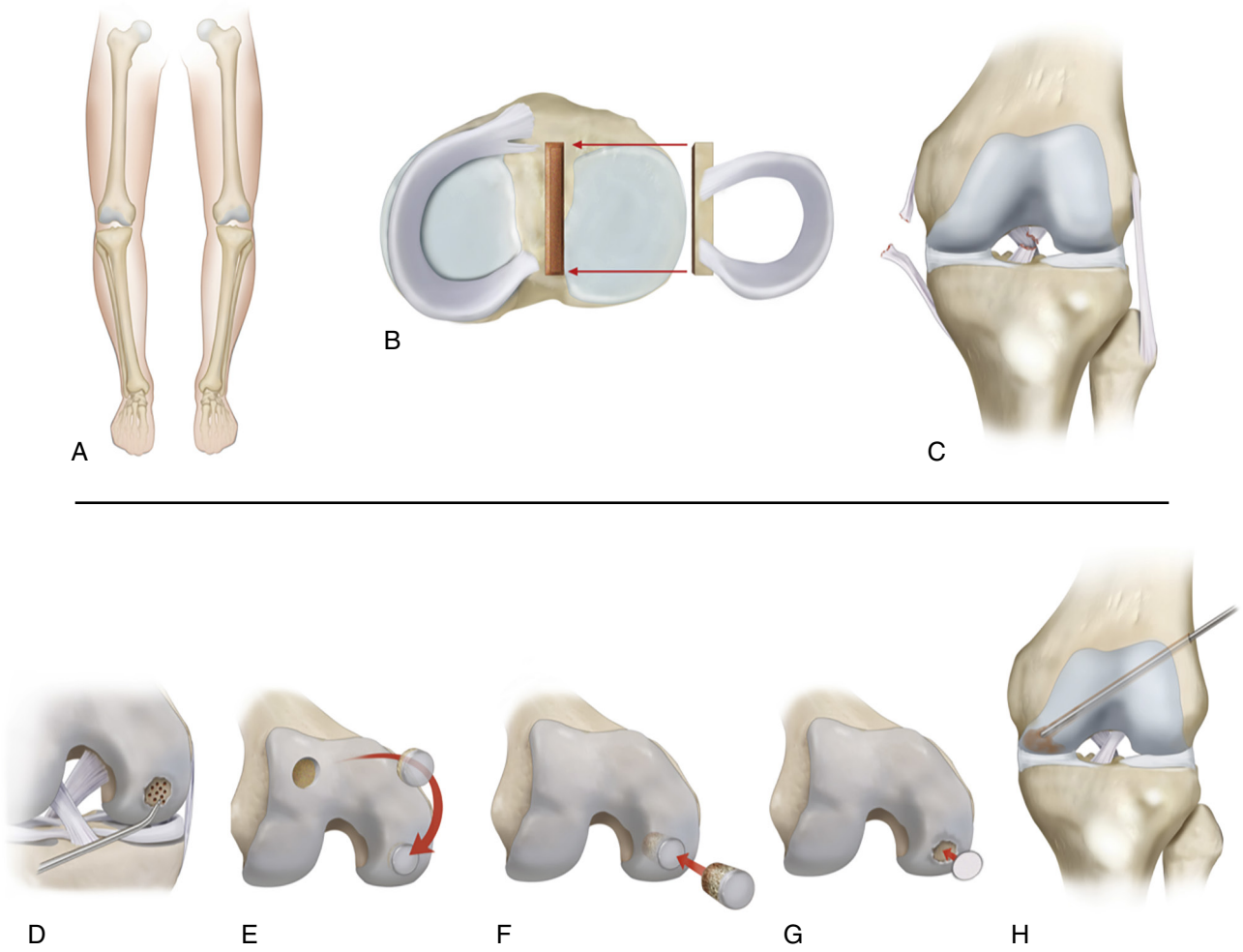
The goal of treating symptomatic FCD is to restore the osteochondral unit in an anatomical fashion while maintaining the supporting subchondral bone and cartilage and minimizing the surgical burden on the patient. The expectation after management of these lesions is pain relief and return to previous level of activity without limitation. The algorithm for treatment of FCD is constantly evolving as different treatment techniques are developed and tested. Before addressing the FCD, the knee requires a comprehensive evaluation with particular attention to extrinsic factors that could contribute to the symptoms or affect the integrity of the planned intervention.

Extrinsic Factors

Extrinsic factors that must be considered include malalignment, concomitant meniscal deficiency, ligament insufficiency and knee instability. If present, these concomitant extrinsic factors can be treated with the appropriate intervention simultaneously (Fig. 22.3): for malalignment, a simultaneous or staged osteotomy (high tibial, distal femoral or tibial tuberosity); for meniscal deficiency, a meniscal repair or meniscal allograft transplantation; for ligament insufficiency and knee instability, a ligament reconstruction or repair, respectively.[34] It is imperative to both identify and address the existence of these pathological conditions because failure to do so will compromise the outcomes of FCD treatment.

Extrinsic Factor I: Malalignment

Malalignment of the tibiofemoral joint can predispose the affected compartment to undue mechanical stress that accelerates the development and progression of intraarticular pathological conditions. If varus malalignment is present in the setting of medial femoral condyle disease, a valgus-producing proximal tibial osteotomy (PTO) should be performed to unload the articular surface and repair the site. Similarly, valgus malalignment can be addressed with a distal femoral osteotomy, a closing wedge PTO (CWPTO) or proximal lateral opening tibial varus osteotomy to off-load the lateral compartment.[35] Failure to correct malalignment has been reported to lead to inferior outcomes after FCD treatment.[36] Moreover, improved functional status and symptom relief have been reported in combined osteotomy and cartilage surgery. Kahlenberg et al.[37] reported on 827 patients who underwent high tibial osteotomies (HTOs) and cartilage repair or restoration surgery with 2-year follow-up and demonstrated improved clinical outcomes with low rates of complications (10.3%). Malalignment should be corrected in conjunction with

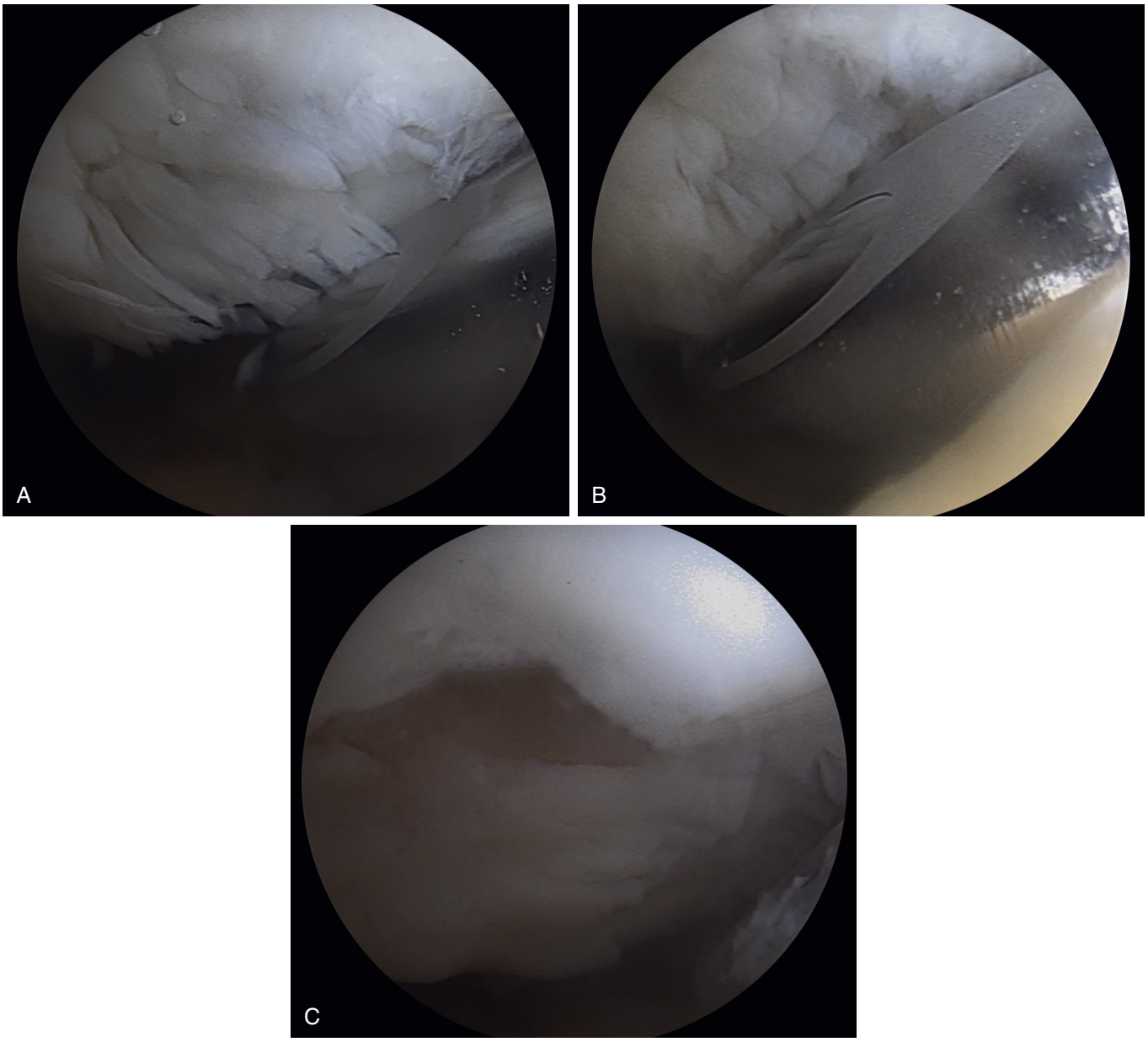


• **Fig. 22.3** Treatment algorithm for focal chondral defect (FCD). Top row illustrates extrinsic pathological conditions and their respective corrections: (A) knee malalignment is addressed concomitantly with a staged or simultaneous osteotomy of the proximal tibia or distal femur. (B) Meniscus deficiency, when not amenable to repair, is treated with a meniscal allograft transplantation. (C) Knee instability is corrected with ligament reconstruction or repair. Bottom row illustrates various treatment options for FCD determined by specific characteristics of lesion: (D) microfracture and (E) osteochondral autograft transplantation are appropriate considerations in smaller FCDs in younger, high-demand patients; (F) osteochondral allograft transplantation and (G) autologous chondrocyte implantation (ACI)/matrix-induced ACI are reserved for larger, deeper lesions, bipolar involvement, or revision surgeries; (H) bone marrow aspirate concentrate is injected into the subchondral defect when the defect is contained within subchondral bone.

FCD treatment to avoid subjecting the treated lesion to inappropriate mechanical stress.

Extrinsic Factor II: Meniscal Pathological Conditions

Cartilage structure and meniscal integrity are closely intertwined. Failure to address either defect during surgery can potentiate the progression of disease. For example, if cartilage procedures are performed in patients who are meniscus deficient, increased contact pressure on the implanted cartilage, graft or developing fibrocartilage may ensue and jeopardise the procedure. In cases where the damaged meniscus is not amenable to repair or has been previously removed in a subtotal meniscectomy, a meniscus allograft transplantation (MAT) in addition to addressing the chondral defect is a viable surgical solution.[38] Success with MAT has been demonstrated with judicious selection criteria; however, meniscal insufficiency in the setting of a FCD, typically on the femoral side, is one of the most challenging pathological conditions to treat. When performed in combination, our preferred technique for addressing the meniscus is through an arthroscopic technique, and the cartilage restoration procedure is then performed using an appropriate technique for the indicated procedure (i.e., arthroscopic for microfracture, OAT or MACI versus open for OCA). A systematic review evaluating six studies including 110 patients at a mean follow-up of 36 months found that combined MAT and cartilage restoration or repair had similar outcomes to isolated cartilage repair as determined by the Lysholm Knee Questionnaire, Knee Injury and Osteoarthritis Outcome Score (KOOS), International Knee Documentation Committee (IKDC), Tegner Activity Scale, Modified Hospital for Special Surgery (HSS) Knee Rating Scale and 36-item Short Form Health Survey (SF-36); however, a higher reoperation rate was observed with the combined procedure. The available clinical



• **Fig. 22.4** Surgical procedure of debridement. (A) Arthroscopic photograph of femoral condyle chondral defect demonstrating unstable cartilage flap. (B) An arthroscopic shaver is used to debride the calcified cartilage layer and create a well-shouldered pocket with surrounding healthy cartilage. (C) Exposed subchondral bone after debridement allows for microvascular thrombus formation and fibrocartilage generation.

studies reporting outcomes after combined meniscus and femoral OCA are encouraging as a viable and predictable joint preservation strategy.[34,39]

Extrinsic Factor III: Ligamentous Insufficiency and Knee Instability

Ligamentous insufficiency and instability of the knee necessitates ligament reconstruction or repair to avoid suboptimal outcomes after FCD treatment. Failure to address concomitant instability or ligamentous insufficiency may result in jeopardisation of the restored chondral surface through abnormal joint kinematics, further osteochondral damage, and predisposition to advanced progression of OA. Accordingly, we recommend performing primary ligamentous reconstruction, addressing the chondral defect and, if only the subchondral surface is jeopardised, adding bone marrow aspirate concentrate (BMAC) (see Fig. 22.3). Addressing ligament deficiencies in addition to FCD has been shown to be safe and efficacious. A retrospective comparative study of 75 patients undergoing OAT who had either anterior cruciate ligament (ACL)-intact or ACL-reconstructed knees demonstrated statistically similar failure rates and clinical outcomes at a minimum of 2 years follow-up.[40]

Furthermore, it has been suggested that multiple extrinsic factors, if present, can be addressed simultaneously with good outcomes. Schuster et al.[41] reported on 23 knees that underwent

combined ACL reconstruction, PTO and chondral abrasion or microfracture and found significant improvements in the IKDC score at 5-year follow-up. They noted that only four ACL grafts were insufficient at final follow-up. Therefore the surgeon should not be hesitant to address all deficiencies to best restore the anatomy and to provide the patient with the best chance for an excellent outcome.

After extrinsic causes of FCD progression have been addressed, attention can be turned to the FCD itself. Generally these management options can be grouped into three categories: palliative (debridement), reparative (marrow stimulation techniques), and restorative (osteochondral grafting, chondrocyte implantation and cellular techniques). All these techniques have been shown to provide therapeutic benefit. The challenge is determining which intervention is most appropriate given the clinical presentation and chondral defect characteristics. This decision-making process requires a patient-specific focus and consideration of multiple factors that often extend beyond the realm of obvious pathological conditions. These include age, body mass index, presentation (weightbearing pain, nonweightbearing pain, swelling, catching, clicking and aggravating manoeuvres such as stair climbing or descending), occupation, risk aversion (willingness to pursue other surgical options should the primary therapy fail), surgical history and compliance with previous interventions.

Specific characteristics of the defect also need to be understood in order to offer the correct treatment options. Size, location, number, depth and geometry are all defect-specific variables that need to be considered before selecting an appropriate intervention. The condition of subchondral bone and surrounding cartilage and the degree of containment should also be noted. The quality of cartilage on the opposing surface is another important factor that is often overlooked. Even minimal articular wear can have implications on the outcome of these interventions. A good understanding of each variable and how they will be addressed will help ensure a good prognosis for the patient.

Patellofemoral lesions can be addressed with simultaneous realignment procedures to unload the patellofemoral compartment and protect the cartilage repair site. Traditional anteromedialisation of the tibial tuberosity is an effective treatment option when the FCD is on the inferolateral aspect of the patellofemoral joint.[42] Medial patellofemoral lesions are treated with a more vertically oriented anteromedialisation or isolated anteriorisation.[42] For a more detailed description of patellofemoral joint disease and treatment options, please refer to the corresponding chapter in this book.

The treatment algorithm for chondral lesions is typically guided by the presence or absence of comorbid extrinsic factors, lesion size, location of the lesion and activity level of the patient. Primary repair is the standard of care for any chondral injury that is amenable to fixation. These include any acute osteochondral fragments and any unstable or in situ OCD lesions. It is essential to fix large fragments (more than 1 cm^2) of the weightbearing portion of the femoral condyles. A primary repair is carried out through several steps: (1) elevation of the unstable fragment; (2) debridement of the fibrous base and possible microfracture using drilling, rather than awls, if necessary to stimulate healing via bone marrow product consolidation; (3) bone grafting of areas of cystic changes or bone loss; and finally (4) rigid fixation of the fragment under compression. The author's preferred technique is to use headless differentially pitched metallic compression screws that are removed after a period of 8 to 10 weeks of protected weightbearing to ensure healing and to prevent the screws from becoming prominent should the osteochondral fragment subside over time. Second-look arthroscopy for hardware removal just before a transition to full weight bearing for tibiofemoral lesions can be used to examine the osteochondral defect and evaluate the success of the procedure. This can help guide future recommendations regarding the timing and extent of return to sport.

When a lesion is not appropriate for primary repair, a treatment algorithm has been developed that relies on a graduated surgical plan, addressing the pathological condition while minimising iatrogenic damage (see Fig. 22.3). The more invasive salvage options are reserved for when these first-line treatments fail.

First-line treatment typically consists of debridement, abrasion arthroplasty or marrow stimulation techniques, the most common being microfracture. Debridement involves an arthroscopically performed technique where unstable damaged articular cartilage is debrided, potentially reducing the biological burden to the joint and reducing mechanical symptoms caused by flaps of articular cartilage. Abrasion arthroplasty, a more extensive debridement of the cartilage defect, can additionally be performed with the intent of exposing the microvasculature of the subchondral bone in order to stimulate fibrocartilage repair. Microfracture largely replaced abrasion chondroplasty and is considered the gold standard for isolated articular lesions smaller than 2 cm^2. This technique is similar to debridement with the addition of subchondral drilling to encourage chondrocyte and bone marrow cell recruitment and repair at the defect site. This technique is minimally invasive, single stage, low cost and technically easier than other treatments.[43] However, the fibrocartilaginous repair tissue that fills the FCD, primarily composed of type I and III collagen with abnormal proteoglycans, lacks the intrinsic biochemical and viscoelastic properties of normal hyaline cartilage.[44] Moreover, the destruction of the subchondral plate carries concern for subchondral cyst formation and devitalisation of subchondral anatomy.[45]

Although microfracture remains the most popular treatment option for small chondral defects, reports suggest that abrasion chondroplasty carries similar outcomes without compromising the subchondral plate.[46] Independent of the specific method used to stimulate fibrocartilage repair, strict adherence to essential principles, including uniform elimination of the calcified layer, creation of vertical walls at the transition of the defect adjacent to the normal articular cartilage and immediate low- or no-load range of motion for a period of 6 to 8 weeks, ensures the greatest likelihood of a successful reduction of symptoms.

Moreover, there appears to be a role for benign neglect in management of these incidental articular cartilage lesions. Ulstein et al.[47] prospectively reported on 5-year outcomes of 368 patients who underwent primary ACL reconstruction who were found to have a concomitant full-thickness cartilage lesion. The authors found no difference in outcomes between chondral defects that were left unaddressed and those treated with debridement or microfracture, supporting the hypothesis that asymptomatic FCDs do not need to be routinely treated.[47]

Defect size

Lesion size and depth are important factors to consider when determining appropriate treatment. High-demand patients with small lesions or patients who have failed marrow stimulation are candidates for OAT. Larger lesions are better addressed with OCA or autologous chondrocyte implantation (ACI), which is commonly used in conjunction with a scaffold, termed *matrix-induced ACI,* because of durability and defect-filling capabilities. ACI/MACI is more appropriate for surface lesions with uninvolved or healthy subchondral

bone, particularly in the patellofemoral joint. This technique does not violate the subchondral bone and does not limit the option for future treatments with other techniques, such as OAT or OCA. The condition of the subchondral plate is important for guiding therapy. If the plate is compromised, OAT or OCA are often indicated because these replace the entire osteochondral unit. The size of the lesion dictates which technique would be most appropriate. For example, larger, deeper lesions are more appropriately addressed with OCA because of lower donor site morbidity relative to OAT.

Defect location

Location of the chondral defect helps dictate treatment as well. Femoral condyle lesions are the most common symptomatic chondral defect in the knee, followed by lesions in the tibial and patellofemoral compartments.[48] OCA consistently allows for reproducible and accurate anatomical restoration when used for femoral condyle lesions. ACI/MACI also has an excellent outcome profile for lesions of the femoral condyle, especially as a first-line restoration technique with healthy subchondral bone. ACI/MACI and newer surface allografts (Cartiform or DeNovo NT) are also used to address lesions of the patellofemoral joint because the varying anatomical surface topography makes structural grafts more difficult to properly position.

The tibial articular surface is a difficult location to treat. A tibial articular lesion identified at the time of articular cartilage repair of the femoral condyle is usually treated with marrow stimulation techniques such as microfracture alone or with biological augmentation (i.e., BioCartilage). Another option for the treatment of these tibial articular chondral defects is OAT placed in a retrograde manner with a cannulated reamer system. For treatment of larger lesions of the tibial plateau with preservation of meniscus, there has been success reported with OCA or tibial resurfacing and concomitant realignment.[49] This is particularly effective in the setting of fracture and development of secondary arthritis, with graft survival rates up to 65% at 15 years.[50]

Surgical Techniques

Several techniques have been developed to address FCD. These can be classified as palliative (debridement with or without abrasion arthroplasty), reparative (microfracture with or without a biological adjunct) or restorative (osteochondral transplantation, osteochondral allografts and MACI).

Debridement

Debridement refers to the smoothing of degenerative cartilage and stabilisation of unstable cartilage flaps commonly seen in FCD. This technique is performed arthroscopically with a set of curettes and shavers (Fig. 22.4). Low suction should be used on the shaver to remove diseased tissue that is resected while preserving intraarticular pressure to limit bleeding.[51] The goal of this procedure is to remove any calcified cartilage within the defect while taking caution to preserve the subchondral bone and healthy surrounding cartilage.

Abrasion Arthroplasty

Abrasion arthroplasty is a palliative technique for treatment of cartilage defects within the knee joint. The technique is classically described as extensive multiple tissue debridement with arthroscopy.[52] The benefits of this technique include simplicity in technique and instrumentation (requiring only a shaver), as well as the ability to combine other interventions to address concomitant pathological conditions, including meniscal defects and misalignment. Similar to other palliative techniques, debridement of tissue does lead to prolific fibrocartilage replacement of inferior quality that is at risk for degeneration over time. However, the fibrocartilage matrix can provide symptomatic relief for a time in an arthritic knee or symptomatic FCD.

Microfracture

Microfracture is a technique using controlled subchondral perforation to allow for recruitment and accumulation of bone marrow elements, such as mesenchymal stem cells and growth factors, within the FCD. The procedure should begin with an examination of the extremity under anaesthesia to confirm full range of motion and rule out concomitant ligamentous laxity. The limb can be placed in a standard leg holder or maintained in an unsupported supine position. A leg holder may provide better access to the extreme flexion surface of the femoral condyle. A routine 10-point diagnostic arthroscopy should be performed with careful examination of the posterior aspects of the medial and lateral femoral condyles. A probe can be used to assess the integrity of the cartilage. If global chondral changes are observed, microfracture should not be performed.

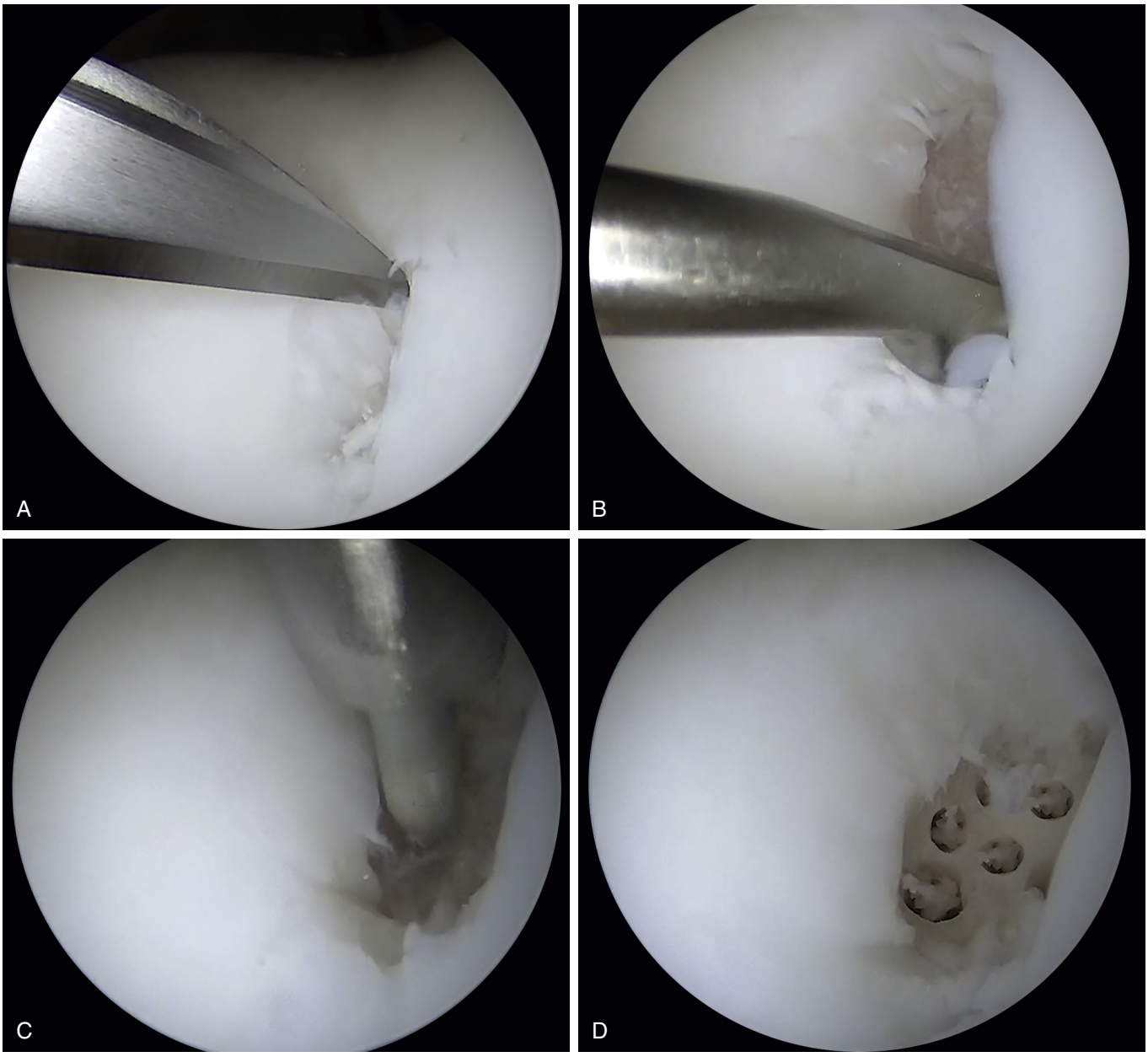
A single FCD or multiple chondral defects can be addressed with microfracture. These lesions should be initially prepared with debridement of the FCD. An arthroscopic blade can be used to sharply debride any unstable cartilage flaps (Fig. 22.5). A curette can then be used to further debride the subchondral shelf and create vertical walls around the cartilage defect, which provide for an area for the clot of marrow product to form and adhere. Additionally, debridement of the calcified cartilage on the base of the lesion may be necessary. This improves adherence of the clot and chondral nutrition through subchondral diffusion.

Historically a surgical awl was used to penetrate the subchondral bone (Fig. 22.6). Concerns regarding subchondral bone insult and inferior repair tissue with microfracture have encouraged the development of alternative, less traumatic techniques. For example, a drill or PowerPick (Arthrex, Inc.) may alternatively be used to create multiple small holes in the exposed bone of the defect (Fig. 22.7). The holes should be made in an outward-in fashion. Additionally, they should be placed 3 to 4 mm apart (three to four holes per square centimetre). To protect the integrity of the subchondral plate, these holes should not connect or be congruent. The periphery of the chondral lesion should be adequately penetrated at the transition zone to stimulate healing of the repair tissue to the surrounding normal articular cartilage. Blood and fat droplets should be seen flowing from the areas of the subchondral penetration at the termination of the procedure.

Alternative techniques such as Nanofracture (Arthrosurface, Franklin, MA, USA) use a smaller diameter and deeper subchondral needling in an attempt to limit the amount of subchondral bone infiltration.[53]

Osteochondral Autograft Transplantation

OAT is a technique that uses a healthy osteochondral plug for replacement of an FCD (Fig. 22.8). Indications for OAT are localised, unipolar, relatively small (less than 12 mm^2), symptomatic chondral lesions of the femoral condyle, tibia, trochlea or patella.



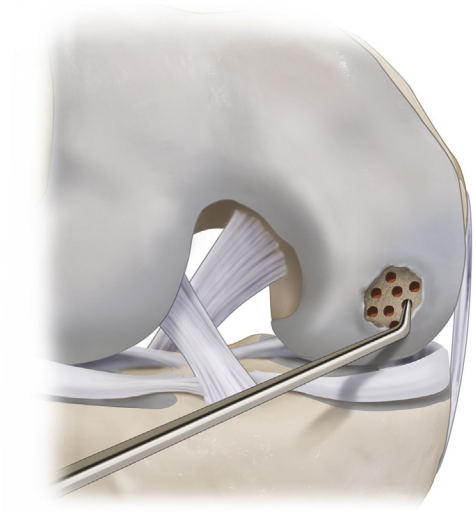
• **Fig. 22.5** Surgical procedure of microfracture. (A) An arthroscopic blade is used to debride the cartilage defect and establish the periphery of the lesion. (B) A curette is used to further debride the calcified cartilage layer and expose subchondral bone. (C) The drill is positioned over the prepared cartilage defect. Subchondral bone can also be punctured with an awl (D). A drill or PowerPick (Arthrex, Inc., Naples, FL, USA) is used to create multiple small holes 3 to 4 mm apart in the defect.

Bipolar FCD of the patellofemoral joint can be addressed with OAT but are more controversial. These patients generally have high physical demands and are younger than 40 years old.

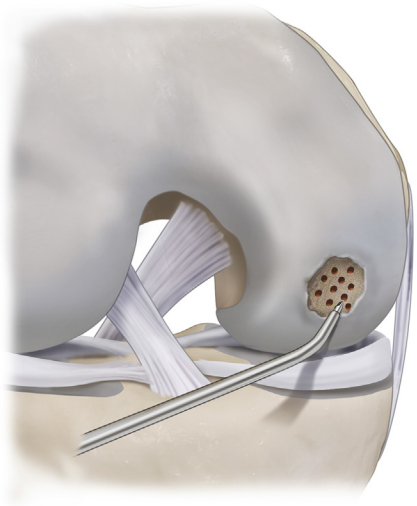
The patient is positioned in either the supine position or the limb can be placed in a standard leg holder to provide a stable, assistant-free knee flexion angle. A tourniquet is often helpful to use throughout the case to maintain haemostasis. The procedure begins with a standard arthroscopic diagnostic evaluation of the knee in which concomitant pathological defects are recognised and addressed. A sizing device is used to determine the size of the FCD. Osteochondral donor plug harvest is then conducted with a corresponding tube extractor inserted at a perpendicular angle to the lateral femoral trochlea just proximal to the sulcus terminalis into the subchondral bone to a depth of 10 to 15 mm with a mallet. The harvester is axially loaded and turned 90 degrees clockwise, then counterclockwise, to preserve the donor plug during extraction.

The recipient site is prepared with the recipient harvester driven perpendicularly into the recipient lesion to a depth of 2 mm less than the depth previously achieved by the donor harvester. Using the same rotational manoeuvres, the recipient plug is removed.

The donor harvester is placed inside the recipient socket and the donor graft is gently extruded. The collard pin of the harvester is advanced until the pin is flush with the pin calibrator and



• **Fig. 22.6** Illustration depicting a surgical awl being used in microfracture procedure to puncture subchondral bone, stimulating fibrocartilage repair with surrounding healthy cartilage.



• **Fig. 22.7** Illustration demonstrating a microfracture drill that has been touted as a less traumatic intervention to stimulate cartilaginous healing.

the graft sits 1 mm outside of the socket. A tamp that is 1 mm larger in diameter than the plug is used to gently advance the plug further until fully seated. Larger defects often require the use of multiple plugs in what is termed the 'snowman technique'. This involves placing and fixing the first plug, then drilling a second recipient site adjacent to, or partially over the first defect. The knee is cycled to make sure the graft is stable.

Osteochondral Allograft Transplantation

OCA is a technique that has been successfully used for primary treatment of a wide spectrum of articular injuries and joint diseases as well as for the salvage of failed cartilage repair (Fig. 22.9). Cadaveric implant eliminates donor site morbidity and allows for treatment of larger lesions, especially those that involve the subchondral plate or bone (more than 2 to 3 cm^2).

This technique is used to treat articular defects of the femoral condyle, trochlea, or patella in young, often higher demand patients. Failure of microfracture or ACI/MACI is not a contraindication, and bone loss can be addressed with OCA.[54] Donor tissue must be size matched to individual patients based on radiographic, computed tomographic (CT), or MRI measurement. The limited window of chondrocyte viability in fresh specimens present challenges in determining the timing and logistics of surgery.

Before the procedure the surgeon should evaluate all aspects of the case and plan for any adjunct procedures that may be required. A small lateral or medial arthrotomy without dislocation of the patella is usually sufficient for smaller defects (Fig. 22.10). For larger defects, patellar subluxation may be required in order to gain adequate exposure to the joint. For patellofemoral lesions or defects on the lateral femoral condyle, a lateral arthrotomy is used, whereas the medial vastus–sparing approach is used for defects in other locations.

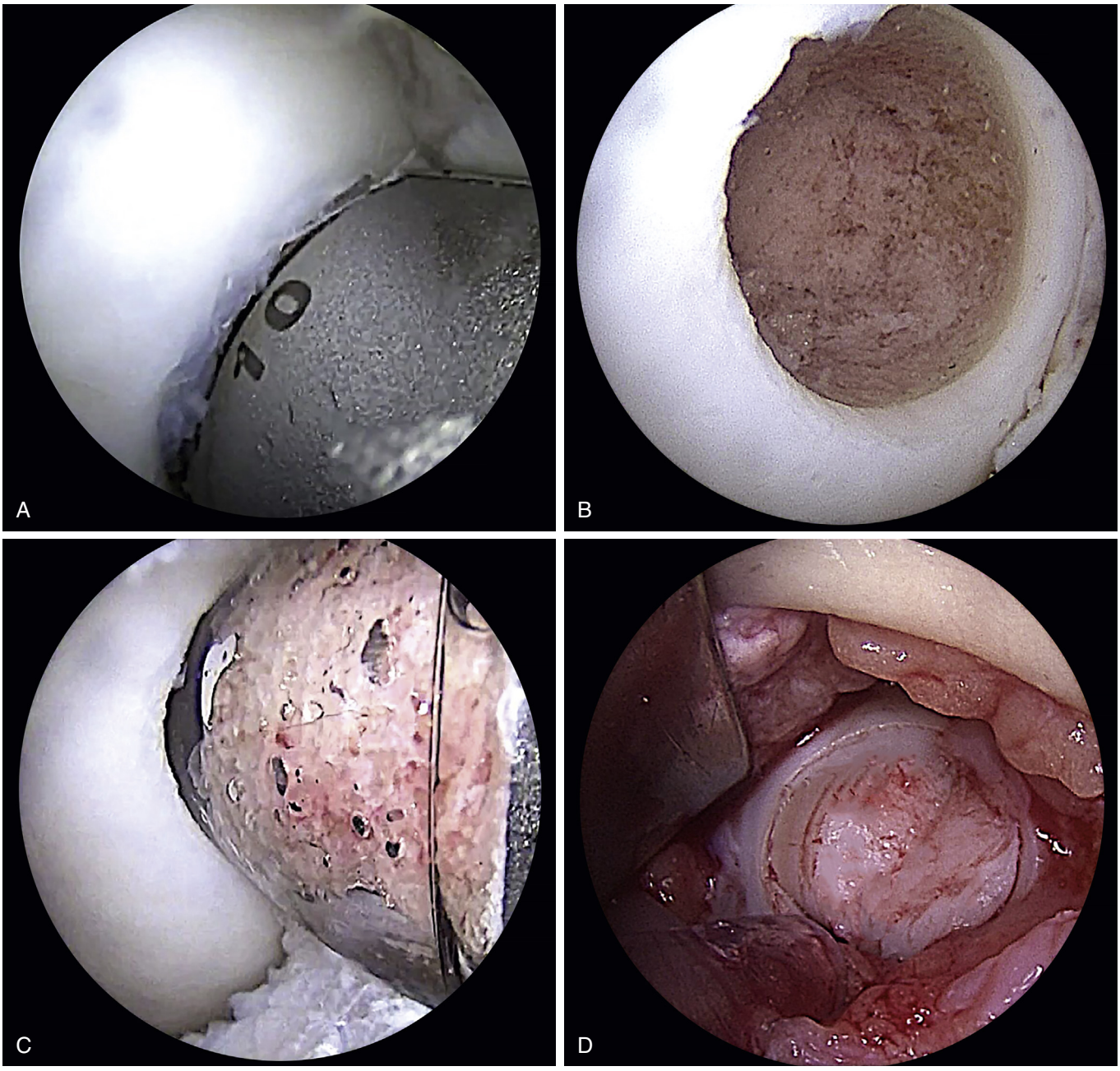
There are two primary surgical techniques: cylindrical press-fit plugs and freehand shell grafts. For the press-fit plug technique, a sizing cylinder is positioned over the lesion. It is always preferable to oversize the reamer and excise the smallest amount of viable tissue necessary than to undersize and leave marginal-quality tissue along the perimeter. Once the appropriate size has been selected, the cannulated sizer is placed centrally over the lesion so that there is complete coverage. Cold irrigation is used throughout the duration of all mechanical steps in an effort to minimise thermal necrosis and preserve surrounding cartilage and underlying bone. The sizer is then placed over the corresponding position on the allograft condyle to make sure a similar-sized plug with comparable anatomy can be harvested. A marking pen is used to mark its location along the 12 o'clock position.

A guidewire is positioned perpendicular to the articular surface into the centre of the lesions using the sizers. The dowel and socket are drilled with a reamer to a depth of 6 to 8 mm. In general, a defect of this thickness (cartilage and bone) is sufficient to avoid transplantation of large amounts of allograft bone. Deeper reaming may be indicated in the setting of osteonecrosis or OCD. Bone grafting may be required in deep lesions and can be collected from the reaming.

The depth of the lesion is carefully measured at the 12, 3, 6 and 9 o'clock positions. The lesion should be cleaned of any frayed cartilage or loose bodies. A calibrated allograft dilator is inserted in the recipient socket and gently tapped to achieve an additional 0.5-mm dilation. When the donor site is sufficiently prepared, attention can be turned to preparation of the allograft. The osteochondral donor plug is harvested from the allograft with the use of a workstation and a coring reamer to match the corresponding recipient site. The measurement made on the recipient site is then transferred on the osteochondral plug, and excessive bone is removed. A corresponding donor harvester is used to drill through the entire donor condyle. Ideally the plug is harvested from a corresponding site on the allograft hemicondyle and the topography is similar to the recipient site. Anatomical landmarks can be used to reference anatomical position on the condyle. These include the sulcus terminalis and the distance to the posterior condyle.

Marking the 12 o'clock position is important to remember graft orientation. The graft is then gently press-fit into the socket, lining up the two 12 o'clock positions on the donor and recipient sides. After the graft is implanted and there is congruity along the joint line, a tamp can be used to gently tap the graft in place.

When tight press-fit cannot be achieved, additional fixation may be indicated. Options for graft fixation include metallic

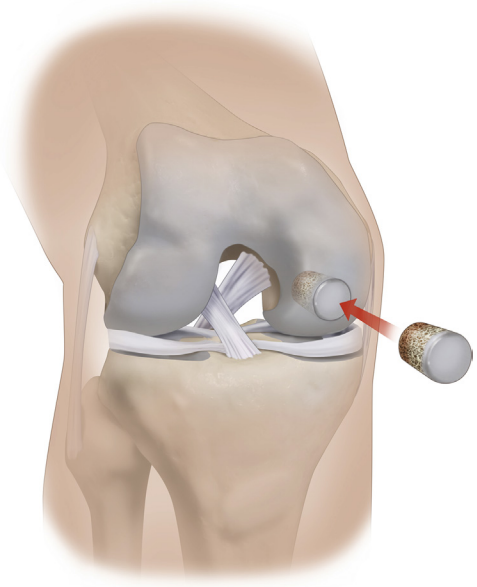


• **Fig. 22.8** Surgical procedure of osteochondral autograft transplantation. (A) The recipient harvester is advanced to a depth of 2 mm less than the depth of the harvested donor osteochondral plug. (B) The recipient socket with a complete and intact rim of surrounding viable cartilage. (C) The harvested plug is inserted coaxially to the recipient socket and should be inserted until seated 2 mm above the intact cartilaginous rim. (D) A tamp 1 mm larger in diameter than the osteochondral plug is used to seat the plug until its borders are congruous with surrounding cartilage.

headless screws, bioabsorbable screws and pins. In cases of larger defects, multiple plugs can be used and placed in the previously described 'snowman' pattern.

Contraindications to the plug technique are lesions that are posterior in the joint. These often cannot be accessed perpendicularly. Tibial plateau defects are also considered exclusionary pathology. In these cases a shell technique can be used. The idea behind this technique is to create a mould of the defect to fill in. Through the use of burr and osteotome, the defect is excavated. A matching shape is formed from donor tissue, which is then fixed with screws and pins. A foil or paper template is used to mould the graft into the same geometric shape as the recipient site. Alternatively, a tibial resurfacing can be performed with an osteochondral allograft by removing 5 to 6 mm of the entire plateau and inserting a size-matched plateau that will be posteriorly fixed with screws on the periphery.[49]

Subclinical immunogenic response after transplantation of donor tissue is a realistic possibility at the bone-to-bone interface; however, the risk of a clinically significant response within the joint is low because of the intact cartilage matrix preventing contact between the donor chondrocytes and host antibodies.[55]

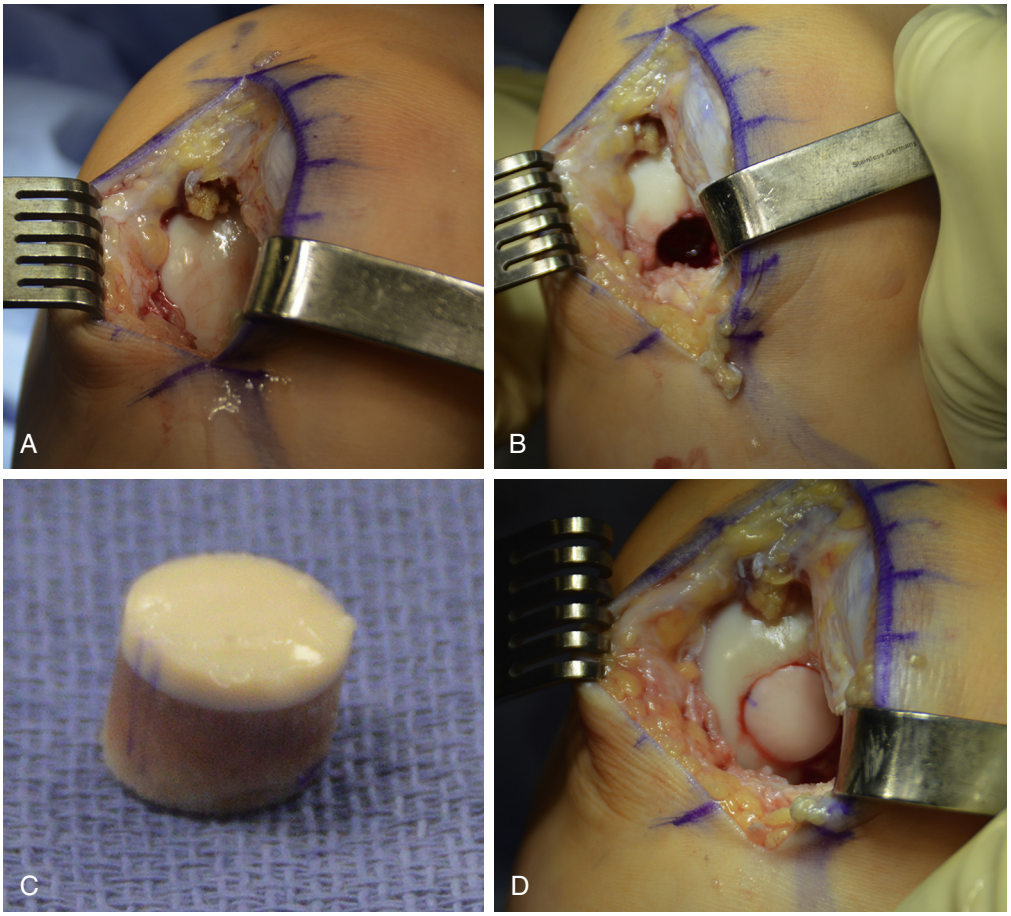


• **Fig. 22.9** Illustration depicting osteochondral allograft transplantation in which an allograft donor osteochondral plug is implanted into a prepared focal chondral defect.

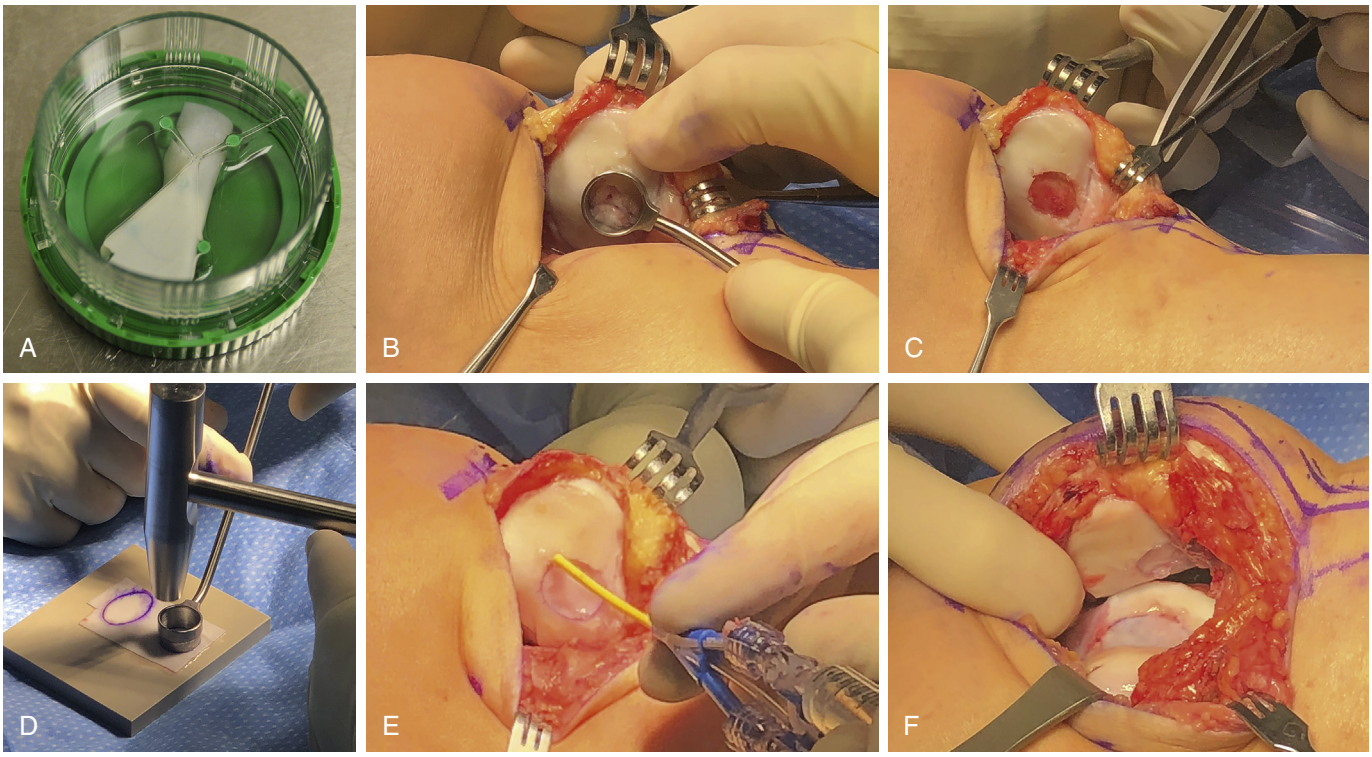
Cellular Techniques

Scaffolds: matrix-induced autologous chondrocyte implantation

MACI is a restorative cartilage treatment technique that touts a stronger construct than its predecessor, ACI, and can be applied in larger FCD. ACI was originally described as a technique that involved harvesting autologous chondrocytes from a nonweightbearing portion of the knee, enzymatically processing said cells and infusing them into a contained defect sealed with either a periosteal patch or synthetic collagen membrane. MACI was developed as a result of concerns regarding the use of chondrocytes in suspension resulting in uneven distribution and possible cell leakage.[56,57] In this technique the culturally expanded chondrocyte cells are embedded on a biodegradable porcine type I/III collagen scaffold (Fig. 22.11). The membrane is then inserted directly into the defect and secured with or without fibrin glue.[56,58] The advantages of this procedure include decreased operating time and surgical exposure because it eliminates sutures or periosteal harvest. Additionally, the scaffold may act as a barrier to fibroblast invasion, which can lead to undesirable fibrous repair.[59]



• **Fig. 22.10** Surgical procedure of osteochondral allograft transplantation. (A) The medial femoral condyle focal chondral defect is properly exposed before site preparation through a medial parapatellar approach. (B) The condyle is reamed perpendicular to a depth of 6 to 8 mm and the defect is explanted, exposing the donor site. (C) The osteochondral donor plug is marked at the 12 o'clock position to correspond with the correct alignment in the donor site. (D) The osteochondral plug is inserted into the medial femoral condyle lesion and tamped down to create a congruous joint surface.



• **Fig. 22.11** Surgical procedure for matrix-induced autologous chondrocyte implantation. (A) Culturally expanded chondrocyte cells embedded on a biodegradable porcine type I/III collagen scaffold. (B) View of a reflected patella with a focal chondral defect demonstrating position of a sizing cylinder over the lesion. (C) View of a reflected patella demonstrating resection of the focal chondral defect. (D) Use of a sizing cylinder to measure the appropriately sized scaffold for insertion into the recipient harvest area of the previous focal chondral defect. (E) Insertion of a biodegradable porcine type I/III collagen scaffold into a focal chondral defect of the patella. This collagen scaffold is secured with fibrin glue. (F) Reduction of the native patella after insertion of the collagen scaffold.

A number of different cell-free scaffolds have since been developed to provide a biological 'net' from which the cartilage can grow into and repair. These three-dimensional constructs are designed to be chondroconductive and osteoconductive. As an adjunct to microfracture, this scaffold provides mechanical stability to the fibrin network of the newly formed blood clot to allow for cartilage regeneration.[60] Some of the more promising technologies available are described next and include third-generation scaffolds (NOVOCART 3D, Aesculp Biologics), minced cartilage procedures (DeNovo NT), viable osteochondral allografts (Cartiform) and extracellular matrix scaffolds (BioCartilage).

Third-generation ACI (MACI analogue or NOVOCART 3D)

NOVOCART 3D is a collagen–chondroitin sulphate scaffold composed of ex vivo expanded autologous chondrocytes seeded on a bioresorbable, biphasic collagen scaffold. This is a two-stage procedure that requires donor site harvesting from a nonweight-bearing portion of the knee joint (e.g., lateral trochlear wall) (Fig. 22.12). The autologous chondrocyte cells are expanded and embedded on a scaffold (Fig. 22.13) that is then implanted in the prepared defect (Fig. 22.14). Although these scaffolds have become popular and show potential in restoring natural cartilaginous matrix in the knee, clinical studies are needed to demonstrate superiority over the already established conservative and reparative techniques.

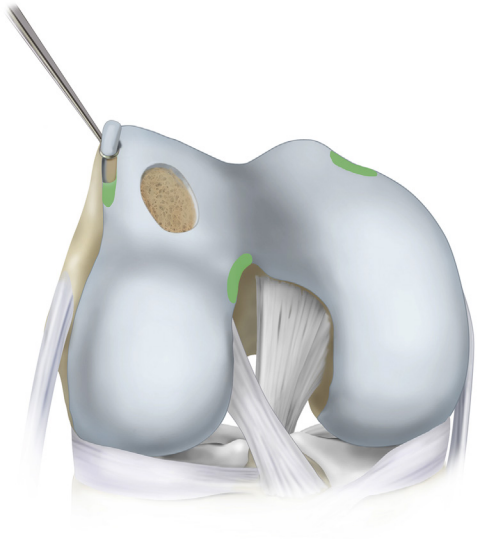
Minced cartilage (DeNovo Natural Tissue)

Minced cartilage is a classic technique originally described in 1983 when Albrecht et al.[61] demonstrated in a rabbit model that particulated cartilage injected into osteochondral defects showed greater healing potential compared with fibrin alone. Moreover, the proliferative nature of implanted chondrocytes allows for a large defect repair with one-tenth of the original amount of cartilage.[62]

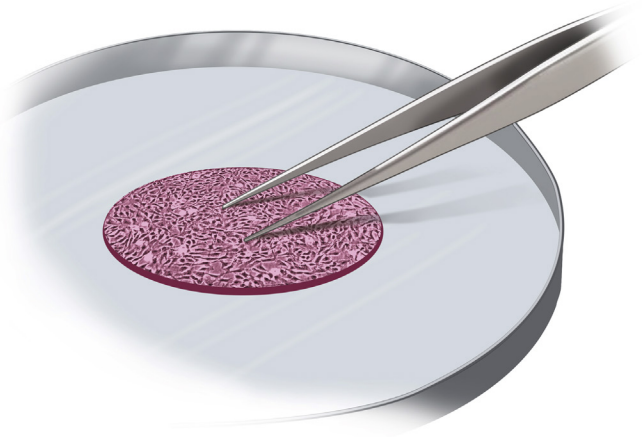
DeNovo NT, one of the leading technologies in this area, is an allograft juvenile articular cartilage minced into 1-mm^3 explants. The cartilage is obtained from the femoral condyle of donors aged from neonates to 13 years. These immature tissues have increased cell density, proliferation rate and outgrowth compared with older donor cartilage.[63] As a viable tissue, DeNovo NT has a limited shelf life of 44 days, so this should be ordered before surgery. The technique for implantation is similar to that of MACI.

The scaffold can be prepared within the defect or extraarticularly. If prepared within the defect, the cartilage pieces should sit approximately 1 mm lower than the surrounding cartilage walls to avoid excessive stress and impact on the implant. The area is then adequately covered in fibrin glue and the knee is flexed to allow for gravity-assisting fibrin placement. After glue adhesion, the knee should be taken through range of motion to ensure stability of the implant.[64]

An extraarticular technique for DeNovo NT application involves creating a negative mould of the defect with a thin piece of sterile foil against the base and walls. DeNovo NT is transferred



• **Fig. 22.12** A biopsy of intact, viable cartilage is taken from a nonweightbearing portion of the knee joint (e.g., lateral trochlear wall).



• **Fig. 22.13** Autologous chondrocyte cells are expanded and embedded into the desired scaffold.

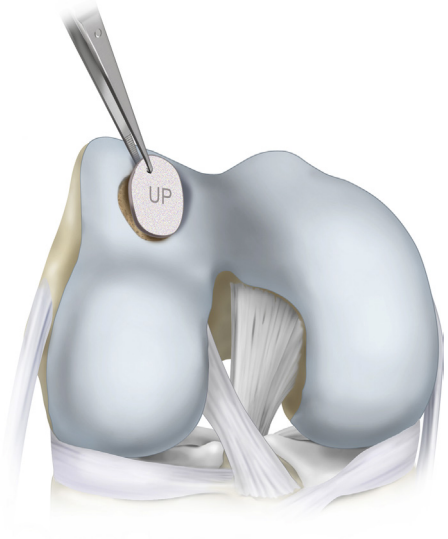
into the mould and then secured with a layer of fibrin glue across the top. After setting, the mould is flipped over and another sheet of fibrin glue is dispersed along the base and sides. The fibrin-fortified implant is then inserted into the lesion and examined to ensure that the implant is not proud on the articular surface. Knee range of motion is inspected for adequate motion and stability.

Viable osteochondral allografts (Cartiform)

Cartiform (Osiris Therapeutics, Inc.) allograft is a cryopreserved viable osteochondral allograft scaffold. The contents of this scaffold include extracellular matrix, viable chondrocytes and chondrogenic growth factors. The graft is cut to match the prepared implant site and then fixed to the bone with fibrin glue with or without sutures or suture anchors.

Extracellular matrix scaffolds (micronized allogeneic cartilage, or BioCartilage)

BioCartilage (Arthrex, Inc.) is desiccated, particulated (100 to 300 μm) allograft articular cartilage scaffold that is hydrated with



• **Fig. 22.14** The matrix-induced autologous chondrocyte implant is cut to the desired size for implantation into the prepared defect.

PRP and placed into contained cartilage defects where microfracture has been performed (Fig. 22.15). This product contains an extracellular matrix that is native to articular cartilage, including type II collagen, proteoglycans and other growth factors. The mixture is injectable after being mixed in a 1:0.8 ratio with PRP and then serves as a scaffold to augment bone marrow stimulation procedures.

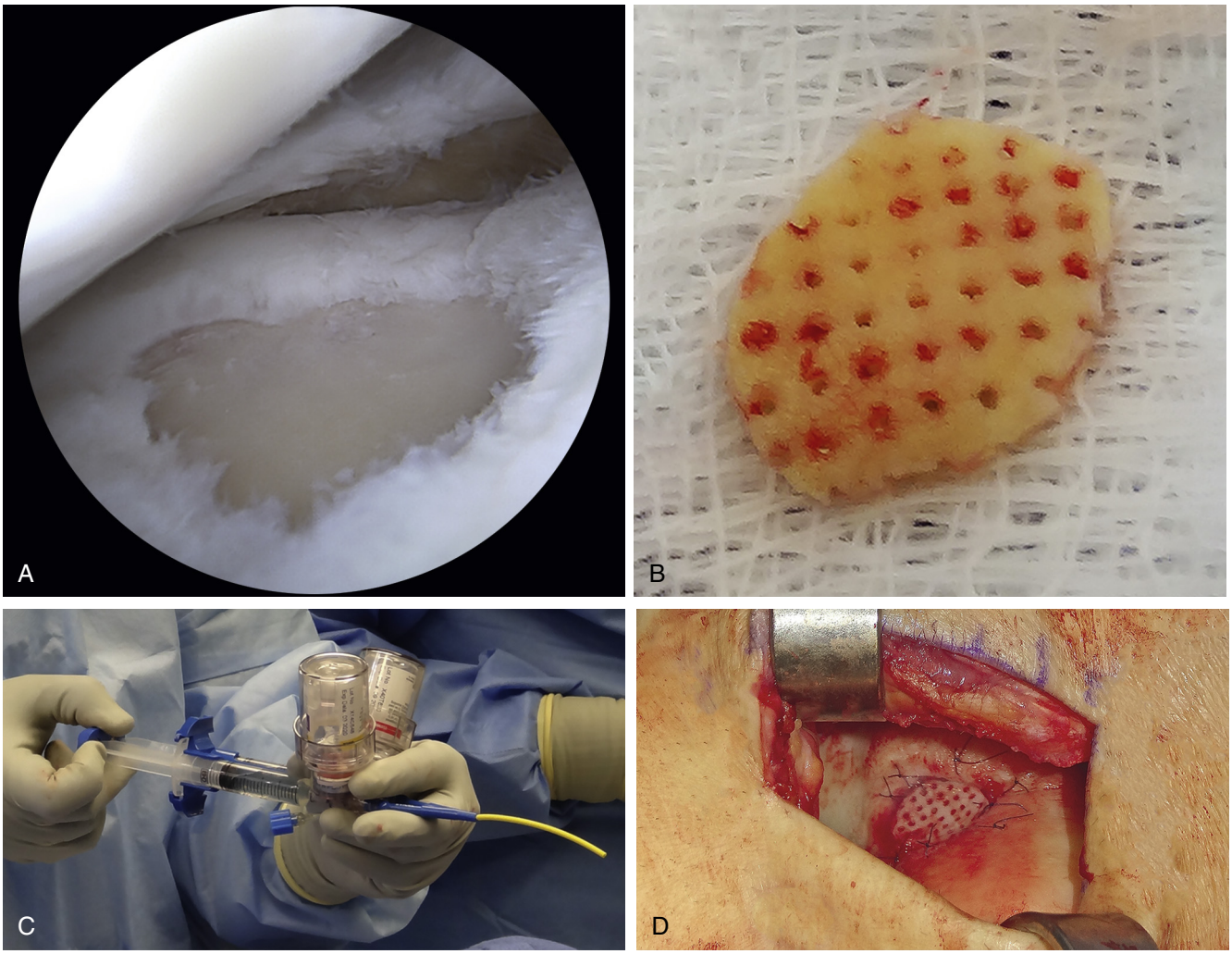
The advantage of these scaffold technologies is the associated low morbidity profile. However, these cell-based techniques are expensive and time consuming and their superiority over microfracture has not been shown in smaller lesions.[65,66] Further research is required to determine whether this technology will become an integral part of FCD management.

Rehabilitation

Postoperative rehabilitation plays a vital role in achieving the best results after treatment of FCD. The rehabilitation protocol varies depending on the location of the FCD. In general the authors recommend after a four-phase protocol for expedited return to function with optimal recovery.

Patellofemoral Lesions

Rehabilitation for patellofemoral lesions should prioritise early full weightbearing, whereas range of motion is more protected early in the postoperative period than rehabilitation for tibiofemoral lesions. Phase I begins postoperative day 1 where patients begin on a continuous passive motion (CPM) machine. The CPM is used 6 hours per day for 6 weeks. At 2 weeks postoperatively, the hinged knee brace is unlocked and permanently discontinued once the patient is able to perform a straight leg raise without an extension lag. We recommend 50% weightbearing in combination with a knee brace during postoperative weeks 0 to 4. Phase I also incorporates physical therapy with an emphasis on passive and active-assisted range of motion. This phase generally lasts 6 weeks.



• **Fig. 22.15** Surgical procedure for biocartilage (Arthrex, Inc.). (A) Arthroscopic view of focal chondral defect on lateral femoral condyle. (B) Desiccated, particulated (100 to 300 μm) allograft articular cartilage scaffold to be placed into area of previous focal chondral defect. (C) The illustrated product contains type II collagen, proteoglycans and other growth factors. The injectable mixture is mixed in a 1:0.8 ratio with platelet-rich plasma, which serves to augment bone marrow stimulation. (D) Insertion of allograft articular cartilage scaffold into focal chondral defect with fixation using sutures.

Phase II spans 6 to 8 weeks, and partial weightbearing is allowed. During this phase, full extension and flexion to 130 degrees should be achieved. Physical therapy in this phase includes quadriceps and hamstring strengthening exercises, in addition to a stationary bike. Phase III spans 8 to 12 weeks, and full weightbearing is permitted in this period. Full weightbearing should typically be achieved by postoperative week 8, at which point exercises are advanced again to include gait training and closed kinetic chain exercises. At 12 weeks, exercise is advanced to include stationary cycling, elliptical training and pool exercises. Full range of motion should be achieved during this time, and physical therapy emphasises closed kinetic chain exercises and restoration of normal gait. Phase IV spans 12 weeks to 6 months and focuses on advanced strengthening with minimal restrictions. Return to sport activity is restricted until after completion of this phase.

Tibiofemoral Lesions

Emphasis for the tibiofemoral group should be range of motion with the use of a brace and patients are advised to use crutches immediately after surgery. Phase I begins postoperative day 1 when patients begin on a CPM machine. The CPM is used 6 hours per day for 6 weeks. At 2 weeks postoperatively the hinged knee brace is unlocked, and it is permanently discontinued once the patient is able to perform a straight leg raise without an extension lag. Depending on the quality of fixation, weightbearing is variable; this may range from complete nonweightbearing status to touchdown weightbearing in this early period. Phase I also incorporates physical therapy with an emphasis on passive and active-assisted range of motion. Phase II begins at postoperative week 6 and partial weightbearing is encouraged. Physical therapy should emphasise quadriceps and hamstring strengthening exercises. Patients should achieve flexion to 120 to 130 degrees during this period. Phase III spans 8 to 12 weeks, and full weightbearing is permitted in this period. Full range of motion should be achieved during this time, and physical therapy emphasises closed kinetic chain exercises and restoration of normal gait. Phase IV spans 12 weeks to 6 months and focuses on advanced strengthening with minimal restrictions. Return to sport activity is restricted until after completion of this phase.

Clinical Outcomes

Microfracture

Although microfracture has long been used as a treatment option for FCD, studies suggest the benefits of the surgery are transient and inferior to the more established restorative treatment options. In a prospective study of microfracture in 110 patients, Solheim et al.[67] found that 50 patients (45%) required conversion to TKA ($N = 7$) and/or had Lysholm score less than 64, indicating a poor outcome. Furthermore, this group found that poor results were more common among patients with mild degenerative changes in the cartilage surrounding the defect, patients with concurrent partial meniscectomy and those with long-standing knee symptoms. Mithoefer et al.[68] performed a systematic review to evaluate the clinical efficacy of microfracture of the knee. The authors incorporated 28 studies focusing on 3122 patients with a minimum of 5-year follow-up and found that microfracture effectively improved knee function in all studies during the first 24 postoperative months; however, after this short-term period the durability of the initial functional improvement was conflicting.

A randomised controlled trial with a minimum follow-up of 15 years sought to compare outcomes between microfracture and mosaicplasty for FCDs of the knee in 20 patients.[69] The authors found that the mean Lysholm score was significantly higher in the mosaicplasty group (67 versus 77, $P = .01$).[69] Chalmers et al.[70] reported that ACI/MACI and OAT demonstrated significant advantages over microfracture with respect to Tegner scores at 1 year and IKDC scores at 2 years. Furthermore, OAT was found to have superior Lysholm scores at 1 year and Marx scores at 2 years. This systematic review suggested that ACI/MACI and OAT purport superior function in the short-term after surgery compared with microfracture.

Osteochondral Allograft Transplantation

OCA has demonstrated good to excellent outcomes in a variety of populations. High rates of return to sport and functional recovery have been observed in recreational,[71] high school and collegiate[72] and elite athletes,[73] and those with a history of previous ACL reconstruction procedures,[40,74] or need for concomitant MAT.[75] Older and obese individuals[76,77] have had successful outcomes.[78] A large database study of cartilage restoration procedures found that in 1608 OCA procedures the reoperation rate was 12.22% at 2 years.[79] A systematic review conducted by Chahla et al.[80] reviewed clinical outcomes and failure rates after OCA of the patellofemoral joint at a minimum of 18 months. The authors determined that the mean survival rate was 87.9% at 5 years and 77.2% at 10 years. Furthermore, the modified d'Aubigné-Postel Score, IKDC, Knee Society Score Functional Component, and Lysholm Knee Questionnaire results all demonstrated mean statistically significant improvements from the pooled study population.

Outcomes after adjacent-plug OCA for both irregular or ovoid lesions and multifocal OCA for multicompartmental, focal defects are now gaining attention because of the complexity of the defects. A retrospective study identified 9 patients (9 knees) who underwent isolated, condylar OCA with the snowman technique and 13 patients (15 knees) who underwent multifocal OCA to quantify survival. This study found a reoperation rate of 44.4% in the snowman group and 20.0% in the multifocal group. Furthermore, there was a failure rate of 33.3% in the snowman group at 7.7 ± 5.5 years and a 6.7% failure rate in the multifocal group at 4.5 years, with all these patients subsequently undergoing arthroplasty. In terms of clinical outcomes, the authors found that patients who underwent snowman OCA demonstrated significant postoperative improvement in KOOS pain subscore and Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) overall score ($P < .05$, both) and that patients who underwent multifocal OCA demonstrated significant improvement in IKDC score; KOOS symptoms, activities of daily living, sport, and quality of life subscores; WOMAC stiffness, function, and overall subscores; and 12-Item Short Form Health Survey physical component (SF-12 physical) summary score ($P < .05$ for all). The authors concluded that patients who underwent unicondylar, multiplug OCA using the snowman technique demonstrated inferior clinical outcomes, higher reoperation rates and greater failure rates than those who underwent isolated single-graft transplantation and that multifocal OCA was a viable technique for knee preservation with multicompartmental chondral disease.[81]

Osteochondral Autologous Transplantation

Many studies have demonstrated excellent outcomes in the postoperative period among patients receiving OAT for FCDs in terms of both clinical improvement and restoration of function. Baltzer et al.[82] assessed short- to midterm outcomes in 112 patients who underwent OAT and found that both the visual analogue scale (VAS) pain (7.14 ± 0.19 versus 3.74 ± 0.26) and WOMAC (134.88 ± 5.84 versus 65.92 ± 5.34) scores significantly improved ($P < .001$ for both) at a mean follow-up of 26.2 ± 0.24 months. A systematic review that examined long-term clinical outcomes in patients who underwent OAT demonstrated significant improvements from baseline in the IKDC and Lysholm scores at a mean 10.2-year follow-up; however, they did not observe significant improvements in the Tegner score.[83] Lynch et al.[84] concluded in a systematic review of 607 patients that patients who underwent OAT had better clinical outcomes and higher rates of return to sport than patients who underwent microfracture. Compared with microfracture, patients who undergo OAT maintain superior levels of athletic activity at intermediate follow-up compared with those who undergo microfracture.[85] A prospective, randomised clinical study with 10-year follow-up found that although patients who underwent OAT and microfracture demonstrated statistically significant improvements at long-term follow-up, patients who underwent OAT had fewer failures (14% versus 38%, $P < .05$).[86]

Pareek et al.[83] in a systematic review of 610 patients who underwent OAT found that the failure rate was 28% and the reoperation rate was 19% at a mean 10.2-year follow-up. The authors also determined that increased age, previous surgery and defect size positively correlated with increased risk of failure, whereas concomitant surgical procedures negatively correlated with failure rate. Riboh et al.[87] in a network meta-analysis created a comprehensive model allowing pairwise comparisons of OAT with other cartilage repair techniques, including microfracture and ACI/MACI, at 2, 5 and 10 years postoperatively. The study ultimately included 19 randomised controlled trials including a total of 855 patients. The authors of this study determined that no differences were observed at 2 years postoperatively; however, at 5 years OAT had a lower reoperation rate than microfracture (odds ratio (OR) 0.03, 95% confidence interval (CI) 0.00 to 0.49), and at 10 years OAT had a lower reoperation

rate than microfracture (OR 0.34, 95% CI 0.12 to 0.92) but a higher reoperation rate than second-generation ACI/MACI (OR 5.81, 95% CI 2.33 to 14.47).

Autologous Chondrocyte Implantation and Matrix-Induced ACI

In the majority of patients, ACI/MACI has been proven to be a safe and efficacious procedure with low complication rates. Indeed, a large database study that included 137 ACI/MACI procedures demonstrated that patients who underwent ACI/MACI had a complication rate of 0.75%.[88] Interestingly, it has been noted that there are higher complications rates with the use of first-generation ACI/MACI compared with second-generation ACI.[89] The four most prevalent postoperative complications after ACI/MACI include hypertrophy of the transplant, disturbed fusion of the regenerative cartilage and healthy surrounding cartilage, insufficient regenerative cartilage and delamination, with periosteum-covered ACI/MACI reported to result in the highest incidence of complications.[90]

In general, ACI/MACI confers good outcomes in both the short and long term. Specific demographic and clinical factors that increase the likelihood of good outcomes after ACI/MACI include younger age and shorter preoperative duration of symptoms;[89] non–worker's compensation cases also had better outcomes.[91] Siebold et al.[92] analysed the outcomes of 30 consecutive patients treated with all-arthroscopic ACI/MACI using chondrospheres for full-size articular cartilage defects at a mean 3-year follow-up. The authors found that 86.6% of patients were completely satisfied with their outcomes and would undergo the procedure again. Furthermore, patients demonstrated significant improvements in Lysholm, KOOS, and IKDC scores and T2 MRI mapping. T2 mapping demonstrated similar cartilage quality of the area of the ACI/MACI compared with the same location at the contralateral knee.[92] In a large case series of 827 patients who underwent implantation with gel-type autologous chondrocyte (Chondron, Sewoon Cellontech Co., Ltd., Seoul, Republic of Korea) or periosteum or matrix-assisted chondrocyte implantation, Nawaz et al.[93] sought to evaluate midterm functional outcomes. At a mean 6.2-year follow-up, all patients demonstrated significant improvements in pain and function. Kaplan-Meier survival analysis revealed that the unadjusted graft survival rate was 78.2% at 5 years and 50.7% at 10 years for the entire cohort. Furthermore, survivorship in the group with a previous cartilage regenerative procedure was inferior to that in patients with a previously untreated lesion, with failure five times more likely in the former group (hazard ratio (HR) 4.718, standard error (SE) 0.742, 95% CI 3.466 to 6.420, $P < .001$).

Third-Generation ACI (MACI Analogue; NOVOCART 3D)

NOVOCART 3D has been shown to result in good clinical outcomes in the medium- and long-term for the treatment of cartilage defects,[94] even in the paediatric and adolescent populations.[95] Studies have also supported that NOVOCART 3D may increase graft maturation. A prospective study concerning MRI with T2 mapping investigated in vivo graft maturation after NOVOCART 3D at a minimum 36-month follow-up and found that T2 relaxation times decreased from 41.6 milliseconds to 30.9 milliseconds at 36 months postoperatively, which were comparable to native hyaline cartilage surrounding the repair. However, the authors failed to find a correlation between the IKDC score and T2 relaxation time values.[96] Niethammer et al.[97] sought to quantify graft hypertrophy after NOVOCART 3D use because it is one of the primary complications of this matrix-based ACI procedure. The authors studied 41 consecutive patients and noted that graft hypertrophy was identified in 11 of these patients by 2-year follow-up and that this occurred more often when the defect was secondary to acute trauma or OCD. In all patients the modified **magnetic resonance observation of cartilage repair tissue (MOCART)** score was significantly improved at latest follow-up compared with baseline. Interestingly, in a subsequent match-paired study using T2-weighted MRI mapping, the authors were able to demonstrate that graft hypertrophy was not correlated with reduced cartilage quality.[98]

Minced Cartilage (DeNovo Natural Tissue)

There is a paucity of literature describing outcomes after use of DeNovo NT (Zimmer Inc.) for treatment of FCD. Farr et al.[99] performed a case series of 25 patients with articular cartilage lesions treated with particulated juvenile articular cartilage (PJAC) and found statistically significant improvements in clinical outcomes as early as 3 months postoperatively. They also found that MRI T2-weighted scores were suggestive of articular cartilage approximating that of normal articular cartilage. Histologically, repair tissue was composed of a mixture of hyaline and fibrocartilage with a high proportion of type II collagen. They noted no reoperations and only one incidence of graft delamination.

Viable Osteochondral Allografts (Cartiform)

Krych et al.[85] sought to evaluate the ability to return to sporting activity after viable osteochondral allografts of the knee in 43 athletes. The authors found that at a mean 2.5-year follow-up, limited return to sport was reported by 88% of patients, while 79% returned to pre-injury level. Furthermore, these authors found that preoperative duration of symptoms longer than 12 months and age over 25 years negatively influenced the ability to return to sport.

Notably, one concern regarding outcomes for FCDs treated with viable osteochondral allografts is whether prolonged storage time affects potential for improvements. Schmidt et al.[100] performed a matched-pair study of patients who received early release grafts ($n = 75$) with a mean storage time of 6.3 days (range, 1 to 14 days) versus those who received late release grafts ($n = 75$) with a mean storage time of 20 days (range, 16 to 28 days). At a mean follow-up of 11.9 years and 7.8 years for the early and late cohorts, respectively, the authors found that failure occurred in 25.3% of early release patients and 12.0% of late release patients. The authors failed to find a difference in postoperative pain and function, and 91% and 93% of early and late release patients, respectively, reported they were satisfied with their outcome, suggesting that prolonged storage is safe and effective. A systematic review of 18 studies that reported viable OCA for cartilage defects of the knee found that storage time ranged from 7 to 43 days. The authors also found that the failure rate of these procedures ranged from 0 to 85.7%, although most studies reported some frequency of failures that ultimately required reoperation.[101]

Extracellular Matrix Scaffolds (Micronized Allogeneic Cartilage, or BioCartilage)

Reports of the outcomes of extracellular matrix scaffolds for the treatment of FCD of the knee are scarce. One controlled laboratory study sought to evaluate the efficacy and safety of BioCartilage in an equine model compared with microfracture.[102] The authors created two 10-mm-diameter full-thickness cartilage defects in the trochlear ridge of five horse of the knees bilaterally. The authors found that the ICRS repair scores in both proximal and distal defects were significantly better in the BioCartilage group compared with the microfracture group (7.4 ± 0.51 versus 4.8 ± 0.1, P = .041) at the time of euthanasia at 13 months. Furthermore, the authors reported that BioCartilage improved histological scores for repair–host integration, base integration and formation of type II collagen compared with positive controls. The authors failed to find a difference on micro-CT analysis; however, using MRI they determined that T2 relaxation was significantly shorter (better) in the superficial region in BioCartilage-treated distal defects compared with microfracture.

Summary

With long-term outcomes only beginning to be reported for these restorative and regenerative cartilage procedures, it will be imperative to continue to describe these outcomes and to better define the indications and treatment algorithm for each. Additionally, there is an important role for basic science studies to improve the integration of graft bone for OCA. OCA mismatch is yet another poorly studied problem, and topographical studies will be necessary to elucidate the extent of mismatch to help the treating surgeon better understand graft sizing and placement, which may increase allograft availability and expand the acceptable allograft pool. With the increase in attention on biological adjuncts in joint preservation procedures, higher-level studies on the use of adjuncts such as PRP and stromal cells will need to be performed to better investigate whether there is a therapeutic benefit conferred with these supplemental treatments.

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