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Cartilage Restoration in the Patellofemoral Joint: Techniques and Outcomes

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Focal chondral defects are common in the patellofemoral joint, particularly in young and active patients, and can significantly impair quality of life. The pathomechanism of cartilage lesions in the patellofemoral joint are multifactorial and can be divided into chronic repetitive microtrauma due to patellar maltracking and acute macrotrauma such as patellar dislocation. Numerous risk factors for patellar instability have been reported including trochlear dysplasia, patella alta, chronic lateral patellar static position, excessive lateral position of the tibial tuberosity, excessive internal rotation of femur/external rotation of tibia, and patholaxity of the medial patellofemoral ligament. Patients with patellofemoral chondral lesions typically present with anterior knee pain that is exacerbated by activity, patellar instability, and/or loss of motion. This is often accompanied by effusion, soft tissue swelling, giving way, crepitance, and loose body sensation. If conservative treatment fails, various surgical treatment options are available ranging from palliative (chondroplasty), over reparative (marrow stimulation techniques) to restorative (autologous chondrocyte implantation and particulated juvenile allograft cartilage), and reconstructive procedures (osteochondral transplantation). A thorough physical and imaging evaluation of the patient is crucial to detect concomitant pathologies and to eventually allow planning a comprehensive individual treatment approach for each patient that addresses both the chondral lesion and any associated pathology. Also, setting realistic expectations and elucidating patient's goals are important parts of a successful treatment. Following these principles, favorable outcomes and high satisfaction rates can be achieved after cartilage restoration in the PFJ.

KEYWORDS Patellofemoral, Cartilage repair, Autologous chondrocyte implantation, Osteochondral allograft transplantation

Background

Focal chondral defects are common in the patellofemoral joint (PFJ) and can significantly impair quality of life. In fact, up to 52% of patients with knee pain or symptomatic osteoarthritis of the knee are diagnosed with chondral lesions in the PFJ when magnetic resonance imaging (MRI) is performed.[1] On arthroscopic evaluation, the PFJ is affected in approximately 30% of the patients that present with grade 3 and 4 focal defects, according to the International Cartilage Repair Society (ICRS) grading, most frequently younger patients.[2,3] The pathomechanism of cartilage lesions in the PFJ is multifactorial and can be divided into chronic repetitive microtrauma due to patellar maltracking (caused by suboptimal extensor mechanism alignment, abnormal osseous structure of the patella and trochlea, loss of integrity of the static and dynamic soft-tissue constraints, and/or generalized ligamentous laxity), and acute macrotrauma, most commonly patellar dislocations, which are caused by either patellar instability or direct trauma.[4,5]

The first step, before defining a specific treatment program for the patient, is to identify how the patient's symptoms relate to the underlying pathology. Osteoarthritis and chondral lesions in the PFJ are commonly associated with symptoms of pain, stiffness, and functional limitations.[6] Thus, symptomatic patients commonly present with anterior knee pain that is exacerbated by activity (typically activities that involve loading of a flexed knee: bending, kneeling, squatting, running, jumping, and using stairs), and functional impairment such as

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patellar instability and/or loss of motion. Secondary symptoms may include effusion, soft tissue swelling, giving way, crepitance and loose body sensation.

Treating cartilage lesions in the PFJ has the goal to increase quality of life by improving pain and function; and ultimately to delay or prevent the potential further need for knee joint replacement.

As most patients present with some form of patellar instability, determining risk factors that contribute to the instability is crucial to guide the treatment planning for these patients. As previously mentioned, recurrent patellar dislocation is a multifactorial problem and the risk of chondral injury is usually related to the amount of energy required to cause patellar dislocation. Normal PFJ anatomy provides both soft tissue and bony constraints to prevent dislocation. Hence, dislocations in these patients are usually the result of high-energy trauma and are consequently associated with chondral or osteochondral injury in up to 70%-96% of patients.[7-10]

Conversely, morphologically abnormal PFJ anatomy, such as trochlear dysplasia, patella alta, chronic lateral patellar static position, excessive lateral position of the tibial tuberosity, excessive internal rotation of the femur/external rotation of the tibia, and patholaxity of the medial patellofemoral ligament (MPFL), puts the patella at increased risk for dislocation, as this environment does not provide sufficient anatomical restraint to prevent dislocation.[11] Patients with one or multiple of these risk factors have a higher risk of dislocation, but the likelihood and extent of articular cartilage damage is much lower compared to high-energy dislocations in anatomically "normal" patients, as less energy is required for patellar dislocation. In order to establish an individualized management plan for each patient, the ideal approach is to systematically identify each pathologic component of the anatomy and plan specific treatment to address each pathologic component.[12,13] Optimization of PFJ stability and biomechanics will ultimately provide the best long-term environment for a cartilage restoration procedure. However, cartilage lesions in the PFJ are particularly challenging to manage due to the complex biomechanics and significant contact stresses that occur during weight-bearing activity.[14]

Before surgical intervention is considered, conservative treatment options that aim to relieve inflammation and controlling pain to regain functional capacity should be exhausted first. This can be achieved with nonsteroidal anti-inflammatory medications, intra-articular corticosteroid injections, hyaluronic acid viscosupplementation[14] in combination with muscle strengthening to improve contact stress distribution across the knee. Also, weight loss and activity modification help to avoid pain aggravation and can lead to improvement in functional limitations.[15,16] If conservative treatment does not result in the desired outcome, surgical intervention is the next step. Generally, operative treatments for chondral lesions can be divided into palliative, reparative, restorative, and reconstructive procedures.[17]

Palliative chondroplasty, which includes lesion debridement and loose body removal, is solely aiming to alleviate mechanical symptoms without providing any cartilage repair. Marrow stimulation techniques (MST) are considered reparative procedures as they stimulate reparative tissue (mostly fibrocartilage) formation in the addressed articular defect.[17] The goal of restorative procedures, represented by first- and second-generation autologous chondrocyte implantation (ACI) and their successor, matrix-associated ACI (MACI), as well as particulated juvenile allograft cartilage (PJAC) is to create a hyaline-like cartilage layer covering the chondral defect. Last, reconstructive procedures include autologous or allogeneic osteochondral grafting, namely osteochondral autograft transfer (OAT) and osteochondral allograft (OCA) transplantation. Both techniques provide fully mature hyaline cartilage and subchondral bone that is transplanted into the defect. Deciding which of these techniques to use depends on defect characteristics such as size, location, and status of the subchondral bone, as well as patient factors such as compliance and resilience, considering the rigorous postoperative rehabilitation process. Risk factors for patellar instability must also be taken into account and addressed if necessary (tibial tubercle osteotomy, MPFL reconstruction, de-rotational osteotomy, etc.). Relative contraindications for cartilage repair generally include smoking, increased BMI, noncompliance, and radiographic evidence of joint space narrowing (Kellgren Lawrence grade III-IV).

Chondroplasty

Chondroplasty refers to debridement and loose body removal with the goal to transform an unstable, irregular chondral lesion into a stable construct. It is frequently performed during the first stage of ACI but can also be indicated as an initial treatment for partial or full-thickness chondral lesions smaller than 1-2 cm^2.[18] For the success of this technique, it is crucial to carefully debride the defect and to avoid transforming a contained into an uncontained lesion, nor to violate the integrity of the subchondral bone.[19] If these principles are followed, good clinical results in terms of pain relieve and improvement of mechanical symptoms can be expected.[20] However, chondroplasty seems to be more successful in traumatic lesions than in chronic, atraumatic chondromalcia.[21]

Marrow Stimulation Techniques

Marrow stimulation techniques include (Pridie) drilling, which utilizes K wires, and microfracture, which utilizes angulated awls.[22,23] Both techniques aim to promote the migration of pluripotent mesenchymal stem cells from the subchondral bone to the chondral defect surface, thereby stimulating fibrocartilage repair.

Due to the distinctive biomechanics and high sheer stresses in the PFJ, and the inferior quality of the repair tissue that is formed by the fibrocartilage, MST is limited to small, contained defects (up to 2 cm^2) in the trochlea in low demand patients that are younger than 40 years with a body mass index (BMI) under 30 kg/m^2.[24]

A meticulous technique is vital to the success of MST. One needs to thoroughly debride down to the subchondral bone, including the layer of calcified cartilage. One must establish vertical and stable walls all around the defect that can sufficiently contain the final repair tissue. Now, perpendicular perforations that are at least 3 mm deep and 3-4 mm spaced apart from each

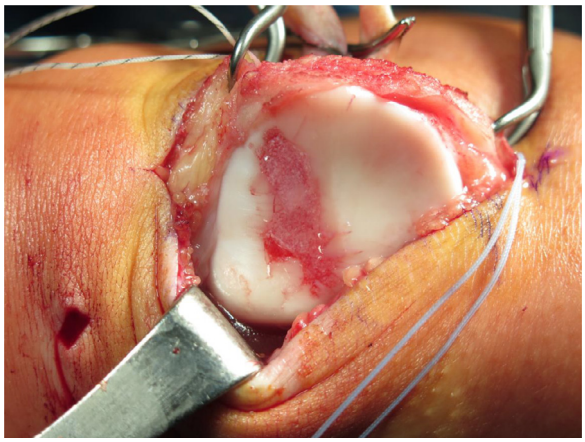

Figure 1 Intraoperative image of a patellar chondral defect that was addressed with marrow stimulation technique (MST) and BioCartilage augmentation, which is sealed with fibrin glue.

other are created in order to promote migration of the pluripotent mesenchymal stem cells into the cartilage defect that ultimately stimulate the production of type I collagen fibrocartilage, without destabilizing the subchondral plate. While trochlea lesions are easily accessible via arthroscopy, patellar defects are more challenging to approach and usually require a small arthrotomy for visualization and proper instrument angulation. For patellar lesions, attention must be paid to apply counter pressure during MST as the greater mobility and increased patellar bone density complicate proper execution.

In general, MST provides short-term improvement that deteriorates over time, with failure rates increasing from 6% at 2 years to 31% at 5 years of follow-up.[25,24] Furthermore, Minas et al[25] and Pestka et al[26] demonstrated the negative effect of MST on the outcome of subsequent cartilage restoration techniques. Prior MST resulted in a significant increase in failure rate of subsequent ACI when compared to primary ACI. Consequently, MST should be cautiously indicated in patients with chondral defects in the PFJ.

Given these limitations of standard MST, increased interest has been devoted to improve outcomes of standard MST by augmenting MST with cell scaffolds, stem cells, and growth factors.[27-30] BioCartilage (Arthrex Inc, Naples, FL) is a cell-based scaffold that comprises desiccated, particulated allograft cartilage that is suspended in platelet-rich plasma. It is placed into the defect after standard MST was performed and sealed with fibrin glue (Fig. 1). This novel scaffold has shown to promote robust hyaline-like cartilage improving results seen after standard MST.[27,31] However, clinical outcomes, particularly in the PFJ, are still pending.

Autologous Chondrocyte Implantation

ACI is frequently performed for the treatment of symptomatic chondral defects in the PFJ, as it allows coverage of larger defects (3-4 cm2) and matching of the complex contour of the patellar and trochlear articular surfaces. While ACI has evolved over time, the 2-stage technique principles remain the same.[32] During the first stage, 100-300 mg of full-thickness articular cartilage are arthroscopically harvested, typically from the superior and lateral margin of the intercondylar notch, and placed in a sterile transport medium for shipment. In vitro, the cartilage matrix is processed and the chondrocytes are cultured for at least 2 weeks but can remain in cryopreservation for up to 5 years. In the second stage, a parapatellar arthrotomy is performed exposing the chondral defect, which is then carefully debrided. The defect is outlined with a fresh scalpel to remove all unstable and altered surrounding cartilage. The surgeon must pay attention not to violate the subchondral plate as an intact subchondral bone is paramount for the success of ACI. Once stable and vertical walls all around the defect are created, the cell product can be implanted. In case of insufficient lesion containment, ACI can be performed in conjunction with OAT to ensure stable surrounding cartilage.[33]

Originally, ACI utilized an autologous tibial periosteal patch to cover the chondral defect, under which the cell suspension was injected and the construct was sealed watertight with fibrin glue to ensure chondrocyte containment within the lesion.[34] Due to issues of periosteal overgrowth with first-generation ACI, the second-generation technique replaced periosteal patches with synthetic or animal-based collagen membranes, which also have the potential of in vivo chondroinduction and chondroconduction[35-37] In the current third-generation ACI, also known as matrix-induced autologous chondrocyte implantation (MACI), the chondrocytes are provided on a pre-seeded scaffold membrane (Fig. 2).[38] The defect is first templated and subsequently a size-matched chondrocyte-seeded collagen membrane is placed in the defect. The cell product is fixed to the defect with either fibrin glue only, or secured to surrounding cartilage with 6-0 absorbable sutures if there is a concern over stability. This new technique significantly simplified the surgery by omitting both the chondrocyte injection underneath a membrane and the watertight sealing.

Patients' medical and surgical history, as well as MR imaging should be carefully evaluated before indicating ACI, as

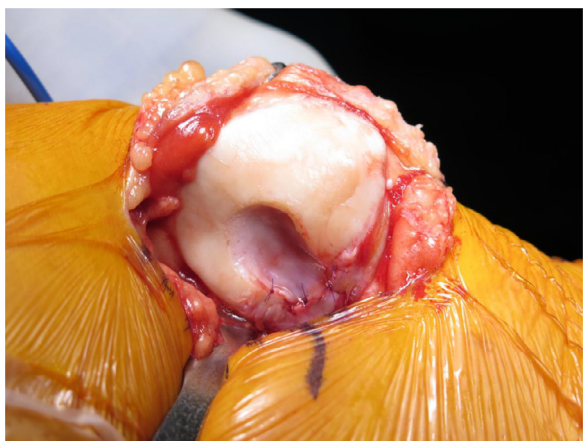

Figure 2 Intraoperative image of matrix-associated autologous chondrocyte implantation (MACI) which is fixed to the defect with fibrin glue. A small rim of degenerated cartilage was left to ensure adequate peripheral containment to which the cell product is secured with 6-0 absorbable sutures, thus providing additional stability.

inflammatory arthritis, obesity, and smoking are considered contraindications for ACI,[39,40] and previous studies reported unfavorable outcomes in patients with prior MST and significant subchondral changes.[25,36]

Clinical outcomes after ACI progressively improved over recent years due to improved techniques and a better understanding of patellofemoral biomechanics.[41-46] In a large multicenter study, ACI to the PFJ resulted in 84% of patients in good or excellent results after a minimum of 4-year follow-up.[5] Isolated patellar defects also show excellent results with a failure rate of 10% after 15 years,[47] especially when lateral facet or inferior pole patellar lesions are addressed with concomitant anteromedializing TTO.[48,49] Newer techniques such as arthroscopic MACI have also shown to result in a high rate (90%) of good or excellent outcomes.[50]

Overall, ACI and MACI have shown excellent results and relatively low failure rates across the knee joint, and particularly in the PFJ.[51,52]

Particulated Juvenile Allograft Cartilage

This cartilage repair technique describes articular cartilage from juvenile donors younger than 13 years old that is mechanically minced into 1-2 mm pieces and stored in vials containing enough material to cover lesions up to 2.5 cm^2 (DeNovo, Zimmer, Warsaw, IN) (Fig. 3).[53] Indications for PJAC include focal full-thickness chondral lesions between 1-6 cm^2 in patients preferably younger than 55 years and a BMI less than 35 kg/m^2.[54]

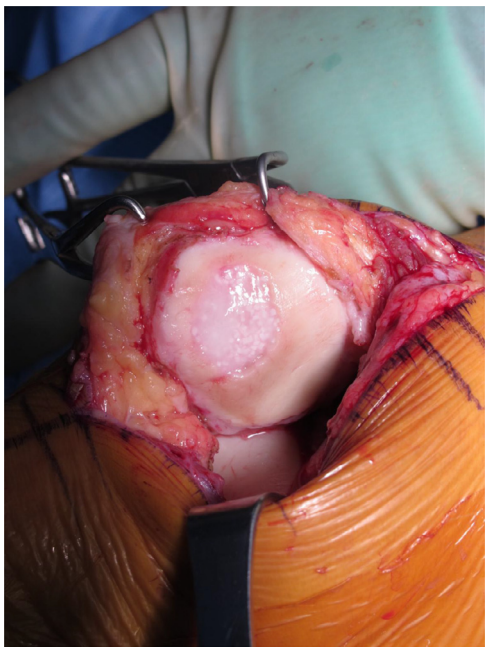

Figure 3 Intraoperative image of a cartilage defect on the patellar articular surface that is filled with particulated juvenile allograft cartilage (PJAC), which is recessed approximately 1 mm below the surrounding shoulders of healthy cartilage before final fixation with fibrin glue to decrease compressive and sheer stresses on the implant.

Analogous to ACI and MACI, the chondral lesion is debrided and stable, vertical walls are created to ensure proper lesion containment. Then, the defect is filled with PJAC, which should be recessed approximately 1 mm below the surrounding shoulders of healthy cartilage before final fixation with fibrin glue to decrease compressive and sheer stresses on the implant. If concerned for stability, or bipolar lesions are addressed, the implant can additionally be covered with a type I/III collagen membrane for protection. While producing a mix of hyaline and fibrocartilage, PJAC has shown to predominately produce type II collagen.[55]

A limited number of reports have been published that investigated the clinical outcomes after PJAC in the PFJ. These reports, however, show encouraging short-term results in patients with focal chondral lesions on the patella and trochlea[55-57] with good to excellent defect fill on postoperative MRI.[55,58]

Osteochondral Autograft Transfer

In this reconstructive technique, 10-15 mm deep osteochondral cylinders are harvested from healthy, nonweight-bearing areas of the knee and are transferred to the defect site of the ipsilateral knee. The advantage of this procedure is the transfer of mature hyaline cartilage with a single-step procedure with good bony integration and no risk of immunologic complications. However, due to remaining concerns about donor site morbidity, OAT has limited indication to only small cartilage lesions up to 2 cm^2 in size.[59] This procedure requires meticulous technique to ensure precise graft fitting and smooth articular surfaces after osteochondral cylinder implantation.

This is particular challenging in the PFJ as the complex anatomy of the patellar and trochlear surfaces complicates creating a smooth articular surface. Additionally, since every donor graft for a patellar lesion will have a thinner cartilage layer than the surrounding healthy cartilage on the patella, local cartilage bone interface mismatch is common.[60] These challenges contribute to the inferior outcomes after OAT seen in the PFJ compared to the femoral condyles.[61] Particularly, patellar lesions show less favorable results with up to 100% failure rate within 1 year postoperatively.[62,63] Yet, other authors showed that OAT is an effective treatment for focal patellar chondral lesions with significant clinical improvement at 2-year follow-up.[64] Similar to ACI in the PFJ, however, concomitant surgical realignment of the extensor mechanism seems to significantly improve clinical outcomes after OAT.[65] Thus, favorable outcomes can be achieved through meticulous implantation of the donor grafts and thorough assessment of associated pathologies.

Osteochondral Allograft Transplantation

This procedure has the same inherent challenges as described for OAT and it cannot be emphasized enough that precise

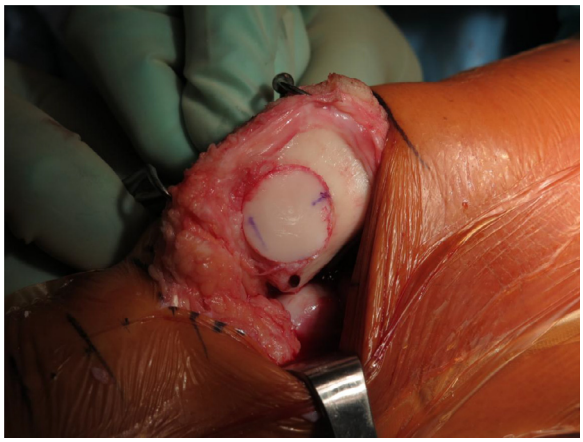

Figure 4 Intraoperative image of a flush-seated osteochondral allograft to the patella. Marking of the 12 o'clock position ensures correct insertion and thus perfect fitting of the osteochondral allograft plug.

fitting of the harvested plug and creating a smooth surface is absolutely vital to the success of both procedures (Fig. 4).

Despite the challenging nature of this technique, OCA has become increasingly popular as a primary or salvage procedure to address chondral defects in the knee, and is typically indicated in osteochondral lesions greater than 2 cm^2.[66,67] The senior author distinguishes between cartilage defects affecting the patellar or trochlear articular surface. In the patella, OCA is usually performed as salvage procedure after failed cartilage repair of lesions greater than 2 cm^2 to provide pain relief and delay arthroplasty in young patients. In the trochlea, OCA can additionally be indicated as a primary treatment option for osteochondral lesions over 2 cm^2.

Currently, fresh osteochondral allografts are stored at a temperature of 4°C for up to 28 days after harvest as this has shown to adequately preserve chondrocyte viability.[68] After the donor plug is harvested in a size and side matched fashion, pulse lavage washing of the graft is performed in an attempt to decrease marrow contents as this is thought to reduce the potential risk of immunoreaction.[69,70] Next, the OCA is precisely fitted into the prepared defect. It should be noted that graft reduction by manual finger compression should be preferred over using a mallet as lower chondrocyte viability after OCA impaction has been reported.[71] Some surgeons augment the OCA plug with bone marrow aspirate concentrate (BMAC) before implantation to promote osseous graft integration.[72]

Compared to OAT, OCA has the advantage of avoiding potential donor site morbidity, and by being a single-stage procedure, OCA is advantageous compared to ACI or MACI. Additionally, as previously mentioned, it can also serve as a revision procedure in patients after failed primary cartilage repair,[68,73] or as a treatment approach for bipolar lesions.[74]

Similar to ACI, smoking and obesity have shown to negatively affect clinical outcomes after OCA and associated pathologies should be simultaneously addressed to achieve improved outcomes.[75]

If accurately performed, OCA transplantation for the treatment of chondral lesions in the PFJ results in excellent clinical outcome with high satisfaction. Transplanting OCAs in the trochlea of 29 knees, Cameron et al[76] reported a graft survivorship of 100% at 5 years and 91.7% at 10 years with improvement of all functional outcomes and an overall satisfaction rate of 89%. Patients with patellar allografts show similar satisfaction rates, but survivorship seems to be inferior with 78.1% at 5 and 10 years, and 55.8% at 15 years.[77] However, conversely to ACI, OCA appears to have higher failure rates in the PFJ compared to the femoral condyles at long-term follow-up (50% vs 24%).[78]

Postoperative Rehabilitation

The following postoperative rehabilitation protocol should serve as a general guideline but it is of utmost importance that each individual patient is carefully evaluated and adjustments to the protocol are made when appropriate.

During the immediate postoperative period, cryotherapy, elevation, and a brace for pain control are recommended, regardless of the specific procedure. During the first 6 weeks, continuous passive motion should be emphasized to avoid arthrofibrosis. After 2 or 3 days, protected weight-bearing is allowed with the knee in a brace for the first 4-6 weeks, followed by progression to full weight-bearing as tolerated. Active training on range of motion is encouraged to further decrease the risk of arthrofibrosis. If concomitant TTO was performed, partial weight-bearing is recommended until 4-6 weeks postoperatively. For reconstructive procedures (OAT and OCA), sport-specific exercises can be started by 4-6 months with return to sport ideally not until 12 months postoperatively. Return to sports in patients who underwent restorative surgery (ACI, MACI, and PJAC) is not advisable until 12-18 months after surgery, depending on the sport (running vs cutting sports).

Conclusion

Cartilage restoration in the PFJ represents a great challenge for orthopaedic surgeons due to its unique anatomy and complex biomechanical environment. Yet, due to improved techniques and better understanding of the PFJ, increasingly good to excellent clinical outcomes and high satisfaction rates can be expected after cartilage repair. Discussion of the patient's current functional limitations is important to define reasonable goals and expectations, and to go over the rehabilitation and recovery time in an attempt to avoid setting unrealistic expectation, which will only lead to disappointment for the patient and the surgeon. Last, a careful physical, as well as imaging evaluation of the knee and lower extremity is of crucial importance to detect concomitant pathology and to allow planning of a comprehensive individualized treatment approach for each patient. Following these principles, favorable outcomes can be achieved that are comparable or only slightly inferior to current cartilage restoration procedures in the tibiofemoral joint (Table 1).

Table 1 Recent Studies That Investigated the Clinical Outcome After Restorative and Reconstructive Cartilage Procedures for the Treatment of Symptomatic Chondral Lesions in the Patellofemoral Joint

Study	Year	Cartilage Repair Technique	No. of Patients	Mean Patient Age in Years (Range)	Mean Follow-Up (Range)	Defect Size, Mean in cm^2	Outcomes
Gomoll[5]	2014	ACI	110	33 (15-55)	90 months (48-192)	5.4	84% good or excellent results, 9 failures (8%) which were treated with partial or total arthroplasty
von Keudell[47]	2017	ACI	30	32 (15-49)	7.3 years (2-15)	4.7	Good to excellent results in 25 (83%) patients, fair in 4 (13%) patients, poor in 1 (3%) patient and 3 failures which were treated with PF and bicompartmental arthroplasty
Ebert[50]	2017	MACI	31	35.3 (16.0-57.0)	5 years final follow-up	2.52	Imaging (MRI) and functional outcome scores significantly improved over time in this prospective study. At 5 years, 80%-93% of patients were satisfied with their outcome
Andriolo[51]	2017	ACI	4294	29 (21-51)	5-12.3 years	4.4	Failure rate of 14.9% (0-42.5) at 86 months. 64.0% of failures happened in the first 12 months, 26.1% between the second and fifth year, and 9.9% after the fifth year
Farr[55]	2014	PJAC	25	37 (18-56)	2 years	2.7	Gradual improvement of all postoperative scores. Mean lesion fill increased from 43.5% at month 3 to 109.7% at 24 months
Buckwalter[56]	2014	PJAC	13	22.5 (14-34)	8.2 months (0.67-32.7)	2.05	Improvement in all KOOS subscores without any complications
Tompkins[57]	2013	PJAC	15	26.4	28.8 months	2.4	Mean postoperative scores: IKDC: 73.3 Kujala: 79 Tegner: 5 VAS pain 1.9 MRI: 73% with normal or nearly normal cartilage repair
Baltzer[62]	2016	OAT	112	48.01	26.2 months	2.55	Improvement in pain and quality of life. Retropatellar defects were associated with poor WOMAC (P= 0.03)
Astur[65]	2014	OAT	33	37.6 (16-59)	30.2 months (24-54)	1-2.5	Significant improvement in Lysholm, Fulkerson and Kujala scores. 83% of plugs had complete osseous integration at 6 months, and 100% at 12 months
Meric[74]	2015	Bipolar OCA	46	40 (15-66)	7 years (2-19.7)	Median, 24.6 (failures) vs. 15.7 (non-failures)	Survivorship was 64.1% at 5 years. Patients with surviving allografts showed significant clinical improvement
Cameron[76]	2016	OCA	28	30.2 (12-47)	7 years (2.1-19.9)	6.1	Graft survivorship was 100% at 5 years and 91.7% at 10 years. 89% of patients were extremely satisfied or satisfied
Gracitelli[77]	2015	OCA	27	33.7 (14-64)	9.7 years (1.8-30.1)	10.1	Survivorship was 78.1% at 5 and 10 years and 55.8% at 15 years. 89% of patients were extremely satisfied or satisfied

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