Lower Extremity-Articular Cartilage Injuries

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Abstract Articular cartilage provides for a smooth low-friction articulation, joint lubrication, and proper stress distribution in order to minimize peak force on the subchondral bone. Acute or repetitive impact can result in articular cartilage lesions, but fortunately in most cases these are asymptomatic. However, when symptomatic, these lesions cause pain, swelling, joint dysfunction, and instability. Multiple non-surgical and surgical therapeutic options are available to treat such chondral injuries. Non-surgical options include oral medications, injections, bracing, and physical therapy. Surgical interventions range from a simple arthroscopic debridement and lavage to allograft transplantation. To determine the proper treatment approach, it is crucial for the operating surgeon to consider the patient's age, symptom intensity, clinical history, post-operative expectations, and lesion characteristics. This chapter provides an overview of the etiology, diagnosis, and management of articular cartilage lesions.

32.1 Epidemiology

Chondral lesions affect approximately one million Americans each year and lead to more than 200,000 surgical procedures for high-grade chondral lesions (grade III or IV) [1]. In a retrospective review conducted by Widuchowski et al., 25,124 arthroscopies identified chondral lesions in 60% of the cases, of which 24% were grade III and 12% grade IV, based on the Outerbridge classification [2, 3]. A similar classification system was used by Curl et al. regarding 31,516 knee arthroscopies. Articular cartilage damage was reported in 63% of patients, of which 60% were grade III and IV [4]. Articular chondral lesions most commonly present in the weight-bearing zone of the medial femoral condyle (32%), followed by the lateral femoral condyle and patellofemoral joint[5].

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32.2 Basic Science

Articular cartilage is avascular and aneural. It is a non-homogeneous tissue, with a complex composition and architecture composed of highly specialized but sparsely distributed cells called chondrocytes. These cells are responsible for the synthesis and secretion of the extracellular matrix (ECM), producing a territorial and inter-territorial matrix composed of water, proteoglycans, and collagen fibrils. Together these cellular and non-cellular elements form the articular cartilage, which maintains joint homeostasis. The articular cartilage is subdivided into four distinct zones:

- The *superficial* (*tangential*) *zone* consists of a fibrillar sheet known as the lamina splendens. It is made of collagen fibers arranged parallel to the surface and a cellular layer composed of flattened chondrocytes. The superficial layer resists shearing stresses, secretes lubricating proteins, and has low fluid permeability. Clinically, it is often the first layer to break down and can be visualized arthroscopically.
- The *transitional zone* is the area of transition between shearing forces and compression forces. It is composed almost entirely of large-diameter collagen fibers and obliquely shaped chondrocytes.
- The *deep radial layer* is composed entirely of collagen fibers arranged perpendicularly to the surface. This zone plays a primary role in load distribution and resistance to compression forces.
- The *calcified layer* is separated from the deep radial layer by the tidemark zone, a transition zone between hyaline cartilage and subchondral bone. This layer contains small cells in a cartilaginous matrix with apatitic salts. Pathologic delamination may occur in this region, which is either preserved (i.e., in cell-based therapy) or intentionally violated (i.e., in marrow stimulation techniques) during cartilage repair procedures.

32.3 Pathophysiology

Articular cartilage lesions can occur acutely (blunt trauma, penetrating injury, or in association with a ligament tear or patellar dislocation) or chronically (long-standing abnormal force distribution across the joint, genetic failure, post-meniscectomy). These lesions can be classified into three types:

- *Partial-thickness injuries* usually occur in the superficial layer and are defined by damage to chondrocytes and ECM components. Such injuries are associated with a reduction in the proteoglycan content and a subsequent increase in hydration, which is strongly correlated with cartilage stiffness. Increased stiffness causes greater load to be transmitted to the collagen-proteolgycan matrix, which accelerates matrix damage and results in the transfer of greater forces to the underlying bone, causing bone remodeling and further breakdown.

- *Full-thickness injuries* present as visible disruptions (fissures, flaps, and fractures) to the articular surface. Such lesions have an inherently poor intrinsic capacity to heal themselves – a characteristic that can be attributed to poor vascular integration and a lack of mesenchymal stem cells. Occasionally, there is mild repair, due to chondrocyte proliferation and matrix synthesis; however, this response is usually short-lived and only partially heals the defect site. Moreover, partial healing ultimately leads to the accelerated degeneration of adjacent articular cartilage due to abnormal load distribution.
- *Osteochondral injuries* are defined by a visible mechanical disruption of the articular cartilage and the subchondral bone. These lesions are commonly observed in adolescents due to the weakness of the calcified zone. Such injuries can occur as a result of acute traumatic events, leading to a fracture that penetrates deep into the subchondral bone, or in response to chronic microtraumatic fractures, as in the case of osteochondritis dissecans. Fractures occurring either acutely or chronically lead to hemorrhage and the formation of a fibrin clot. An inflammatory response is then induced that results in the release of vasoactive mediators and growth factors, both of which stimulate the formation of repair tissue. The repaired tissue is a mixture of hyaline (normal) cartilage and fibrocartilage, with poor stiffness and higher permeability than normal cartilage. Repair tissue rarely persists and over time shows evidence of proteoglycan depletion, increased hydration, and fragmentation. These biologic osteochondral lesions can be appreciated by magnetic resonance imaging (MRI) and do not necessarily require macroscopic or arthroscopic evaluation. Such lesions may behave different clinically compared to full-thickness cartilage damage.

32.4 Classification

Over the years, several classification systems have been published to grade and categorize cartilage lesions according to surface description, diameter, and lesion site [5]. The two most commonly used classification techniques today are the modified Outerbridge and the International Cartilage Repair Society (ICRS) systems [3] (Table 32.1). Both systems are divided into five categories, and the lesions are graded based on diameter and depth. The two systems are similar, but the ICRS allow for more precise classification of lesion grade, region, and dimensions [6].

Table 32.1 Lesions classifications

Lesion grade	Modified Outerbridge classification	International Cartilage Repair Society (ICRS) classification
0	**Normal**	**Normal**
I	Cartilage softening and swelling	Nearly normal: superficial fissuring A: Soft indentation B: Superficial fissured and cracks
II	Partial-thickness defect (< 50% loss of cartilage thickness)	Abnormal: lesion extending down to < 50% of the cartilage depth
III	Fissuring to level of subchondral bone (> 50% loss of cartilage thickness	Severely abnormal: cartilage defect A: Extending down >50% of the cartilage depth B: Down to calcified layer C: Down to but not through the subchondral bone D: Presence of blisters
IV	Exposed subchondral bone	Severely abnormal: penetrating subchondral bone A: Penetrating subchondral bone but not full diameter B: Penetrating subchondral bone and full diameter

32.5 Diagnosis

32.5.1 History and Physical Examination

The evaluation of cartilage injuries starts with a thorough history, including a discussion of the patient's pain and its onset (insidious or traumatic), the mechanism of injury, previous injuries/surgical intervention, and symptom-provoking activities.

Pain is most often the primary complaint and is usually described at the associated compartment: at the medial or lateral joint line for condylar injury and the anterior joint line for trochlear or patellar lesions. Pain associated with chondral lesions may be aggravated by certain positions or weight-bearing activities, whereas activities such as climbing stairs or squatting can aggravate pain associated with lesions in the patellofemoral joint. Pain is usually accompanied by joint effusion, which occurs in the same location and is noted during activity.

A comprehensive musculoskeletal examination should follow to assess for concurrent pathologies such as varus/valgus alignment, patellofemoral malalignment, ligamentous instability, and meniscal deficiency. Range of motion is usually normal in a patient presenting with isolated focal chondral defects; however, ambulation evaluation will demonstrate adaptive gait patterns that shift the weight away from the area of lesion,

such as in-toeing, out-toeing, or flexed knee. Meniscal deficiencies are difficult to differentiate as the associated pain is also caused by articular cartilage lesions. In this scenario, a previous history of meniscectomy can help guide the surgeon towards the possibility of a meniscal deficiency and a plausible cause of continued pain and disability.

32.5.2 Radiologic Evaluation

Standard radiographs remain the most effective tool for initial evaluation of the joint. Radiographic images typically include a weight-bearing anterior to posterior view with the knee in full extension, a weight-bearing 45° flexion posterior to anterior view, a non-weight-bearing 45° flexion lateral view, and an axial view (Merchant view) of the patellofemoral joint. These views enable assessment of the joint space, subchondral sclerosis, and osteophyte and/or cyst formation. In addition, limb alignment, the presence of loose bodies, and osteochondral fractures can be determined.

Importantly, X-rays cannot be used to image cartilage, due to the lack of mineralization. Therefore, patients presenting with continued discomfort after initial assessment are recommended for MRI. Essential information concerning the articular cartilage can be obtained with MRI, for example, regarding the size and depth of the cartilage lesions. MRI also allows for detailed imaging of the subchondral bone, knee ligaments, and menisci and thus helps to determine the presence of concomitant pathologies. Unfortunately, MRI fails to detect some cartilage injuries and often the degree of abnormality tends to be underestimated. Thus, for a patient presenting with unrelenting pain and discomfort, chondral assessment should be made using arthroscopic techniques, considered to be the gold standard.

32.6 Treatment

32.6.1 Non-surgical

Non-surgical management is indicated in low-demand patients, those who prefer to avoid or delay surgical intervention, and patients with advanced degenerative osteoarthritis (a contraindication for articular cartilage restoration procedures).

Oral medications are frequently prescribed in the non-surgical treatment of chondral lesions and include a combination of NSAIDs and oral chondroprotective agents, such as glucosamine or chondroitin sulfate [7, 8]. Glucosamine, an amino sugar, stimulates chondrocytes and synoviocytes to increase ECM production. Chondroitin, a carbohydrate, promotes water retention and elasticity and inhibits fibrin clot formation and, in turn, degradative enzymes. Recent studies have shown improved pain management, increased range of motion, and faster walking speed for patients using chondroprotective agents [9], but there is a lack of clinical data confirming that oral agents affect the formation of articular cartilage.

Other non-surgical options include physical therapy, weight loss, and intra-articular injections with corticosteroids (methylprednisolone or triamcinolone with a local anesthetic) or high-molecular-weight hyaluronans. Recent studies have shown that intra-articular corticosteroids reduce pain for at least one week and should be considered for short-term treatment [10]. On the other hand, intra-articular injections containing high-molecular-weight hyaluronans provide viscosupplementation, which leads to pain reduction and improved joint function [11, 12].

32.6.2 Surgical Options

While the natural history of isolated chondral and osteochondral defects is not predictable, clinical experience suggests that, when left untreated, these lesions do not heal and may progress to symptomatic degeneration of the joint. Therefore, early surgical intervention for symptomatic lesions is often suggested in an effort to restore normal joint congruity and pressure distribution. The goals of surgical treatment are to provide pain relief and improve joint function, allowing patients to return to their daily activities and possibly engage in higher levels of activity.

In general, surgical options can be palliative, reparative, or restorative. The appropriate choice for any cartilage lesion is patient- and defect-specific. The size, location, and depth of the lesion – as well as the physical demands and subsequent treatment options available in case of failure – are variables that must be considered by the operating surgeon (Fig. 32.1). The least destructive and least invasive treatment option that will alleviate symptoms and restore joint function is considered as the first-line treatment.

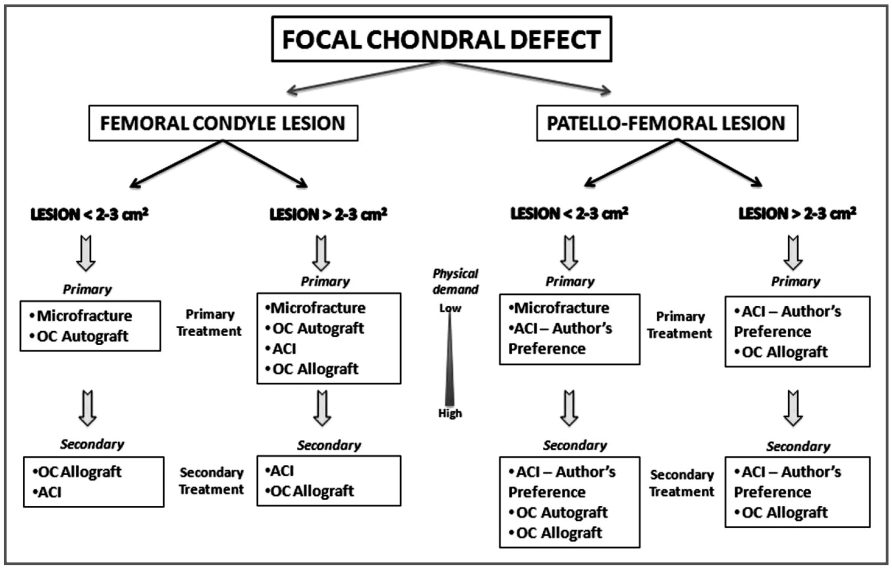

Fig. 32.1 Treatment algorithm

32.6.2.1 Palliative Treatment

Typically, palliative treatments include arthroscopic debridement and lavage. They are reserved for low-demand older patients with small lesions (< 2 cm^2) and limited symptoms [13]. Palliative treatment removes debris, inflammatory cytokines, and proteases (which play a contributing role in cartilage breakdown), temporarily alleviating patient's symptoms and allowing for arthroscopic chondral evaluation. Fond et al., in a cohort observations study, reported successful post-operative outcomes in 88% of the patients at 2 years and 69% of the patients at 5 years, using the HSS scoring system [14]. The recovery time for this type of procedure is relatively short, with weight-bearing and strengthening exercises as tolerated. Many patients who have concomitant meniscal pathology with degenerative arthritis may benefit from arthroscopy and debridement as long as the meniscal pathology is the primary source of joint pain. However, patients must be appropriately educated as to the potential persistence or return of symptoms due to their articular cartilage pathology and thus the need for additional treatment.

32.6.2.2 Reparative Treatments

The most common reparative treatment involves surgical penetration of the subchondral bone, allowing for migration of marrow elements (mesenchymal cells) and the formation of a surgically induced fibrin clot that subsequently results in the production of fibrocartilage at the defect site [15]. This technique of marrow stimulation is applied to several procedures, such as microfracture, subchondral drilling, and abrasion arthroplasty, which are recommended for active patients presenting with small lesions (< 2-4 cm^2) and moderate symptoms. Post-operative outcomes are dependent on physical demand and the inherent regenerative capacity within the joint [16].

Microfracture is the authors' preferred marrow stimulation technique for condylar lesions due to the minimal generation of thermal energy compared to drilling and arthroplasty. The procedure is performed arthroscopically and involves full-thickness cartilage removal down to the subchondral bone and the establishment of well-defined sharp vertical boundaries of normal cartilage to prevent injury propagation. A curette and shaver are used in a single direction through varied portals to facilitate defect preparation. A surgical awl is used to create a bed of small perpendicular holes placed 2-3 mm apart. These holes in the subchondral bone allow mesenchymal cells from bone marrow to enter the defect site and therefore the formation of a surgically induced clot (Fig. 32.2). The clot contains pluripotent cells that have the ability to differentiate into fibrocartilage-producing cells, which fill the defect site with fibrocartilage [15].

For femoral condyle lesions, the post-operative rehabilitation typically involves up to 6 weeks of non-weight bearing or partial-weight bearing and the use of a continuous passive motion (CPM) machine for 6 hours per day. In a patient presenting with a lesion in the patellofemoral joint, weight-bearing is permitted but he or she is advised to wear a brace with a flexion stop at 30° in order to limit patellofemoral contact. Young and athletic patients

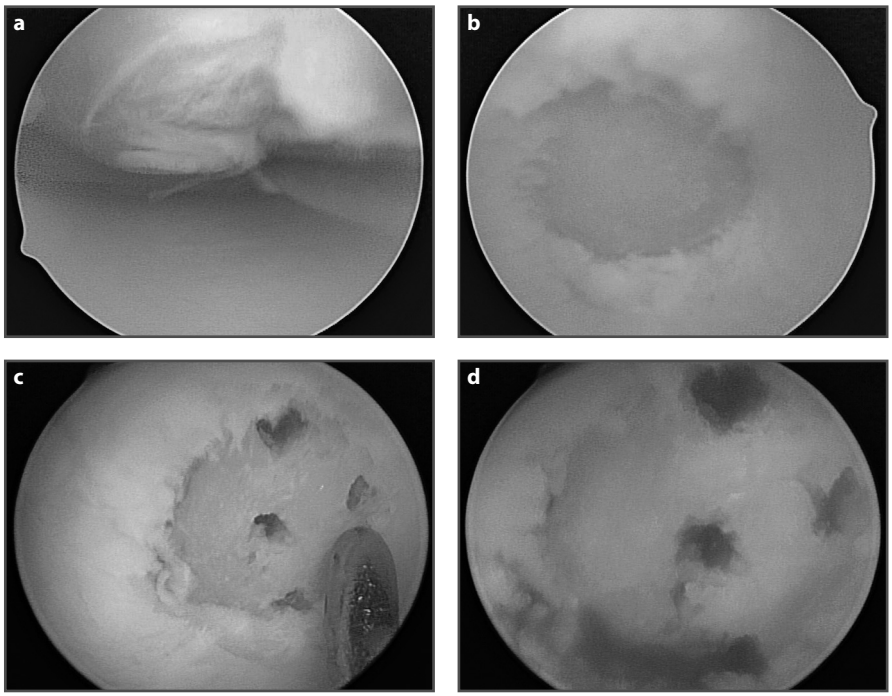

Fig. 32.2 Marrow stimulation technique: microfracture. (**a**) Focal cartilage defect; (**b**) removal of diseased cartilage and formation of vertical wall around the lesion; (**c**) a sharp awl is used to perforate the subchondral bone; (**d**) leakage of pluripotent marrow elements, which will form a fibrin clot and result in fibrocartilage

treated with microfracture for small lesions generally report higher outcome scores than patients with large lesions [17]. Overall, most clinical studies evaluating the outcome of microfracture have reported improvement in knee function in 70-90% of the patients [18]. Long-term results vary, with 60-75% of patients reporting reductions in symptoms and improvement in function [15]. Table 32.2 summarizes outcomes studies for microfracture.

32.6.2.3 Restorative Treatments

The options for restorative treatment include autologous chondrocyte implantation (ACI) and osteochondral auto- or allograft transplantation. ACI is indicated for symptomatic, focal, well-contained chondral or osteochondral lesions measuring between 2 and 10 cm^2, with an intact bone bed. It is the preferred treatment for intermediate to high-demand patients who have previously failed an arthroscopic debridement or microfracture approach [16]. Patients with a patellofemoral lesion and concomitant malalignment should simultaneously undergo ACI and a realignment procedure (anteromedialization of the tibial

Table 32.2 Clinical outcomes of microfractures

Authors	Study group	Lesion characteristics and site	Outcomes	Comments
Knutsen G et al. [19]	Microfracture: 40 patients ACI:40 patients Five-year follow-up	89% MFC 11% LFC	Nine failures (23%) Significant clinical improvement according to Lysholm, SF-36, ICRS, and Tegner scores Better results in younger patients	No significant difference in the clinical and radiographic results between the two treatment groups No correlation between histological quality and clinical outcome Radiographic evidence of early osteoarthritis in 1/3 of the patients in both groups
Solheim et al. [20]	110 patients: single and multiple lesions) Median age 38 years (15-60) Median follow-up 5 years (2-9)	62 MFC 18 trochlea 11 lateral tibia 10 patella 9 LFC Median lesion size 4 cm^2 (1-15 cm^2)	24 failures (22%): 18% in the single-defect subgroup and 29% in the multiple-defects subgroup Improvement in Lymsholm (from 51 to 71) and VAS scores	No significant difference in the Lysholm score between the two groups Significantly lower pain score in the single-lesion group
Steadman et al. [21]	75 cases Mean age 30.4 years (13-45) Mean follow-up 11.3 years (7-17 years)	Traumatic full-thickness chondral lesions Mean lesion size 2.7 cm^2	2 failures Improvement in Lysholm (from 59 to 89) and Tegner (from 3 to 6) scores Good/excellent results according to SF-36 and WOMAC scores	

tubercle) [22]. We recommend the liberal use of a tibial tubercle osteotomy for patients with patellar lesions and/or for central and lateral trochlear lesions. The obliquity of the osteotomy, based upon the trochlear groove to tibial tubercle distance, must be carefully considered so as to avoid excessive medialization of the tubercle, which may lead to abnormal mechanics across the patellofemoral joint.

The first of the two stages of an ACI consists of an arthroscopic biopsy of normal articular cartilage (200-300 mg) from a non-weight-bearing region (intercondylar notch or upper medial femoral condyle), with the sample used for in vitro cartilage de-dif-

ferentiation and culture. The second stage of the procedure, usually done no sooner than 6 weeks after the biopsy, involves a limited arthrotomy to expose the lesion, which is then debrided using a number 15 blade and a sharp ring curette to form vertical walls of normal articular cartilage. Next, a synthetic patch (off-label usage, Bio-Gide, Geistlich Biomaterials, Wolhusen, Switzerland) is sewn at the lesion site using 6-0 Vicryl (polyglactin) and sealed using fibrin glue. The use of a collagen membrane as shown to decrease the re-operation rate for hypertrophy from 25.7% to 5% [23]. All edges of the patch are sealed using fibrin glue, except for a gap at the upper edge, which should be maintained to allow for chondrocyte implantation. Cultured chondrocytes are delivered through the gap using an angiocatheter and, once implanted, the gap is sealed using suture and fibrin glue (Fig. 32.3).

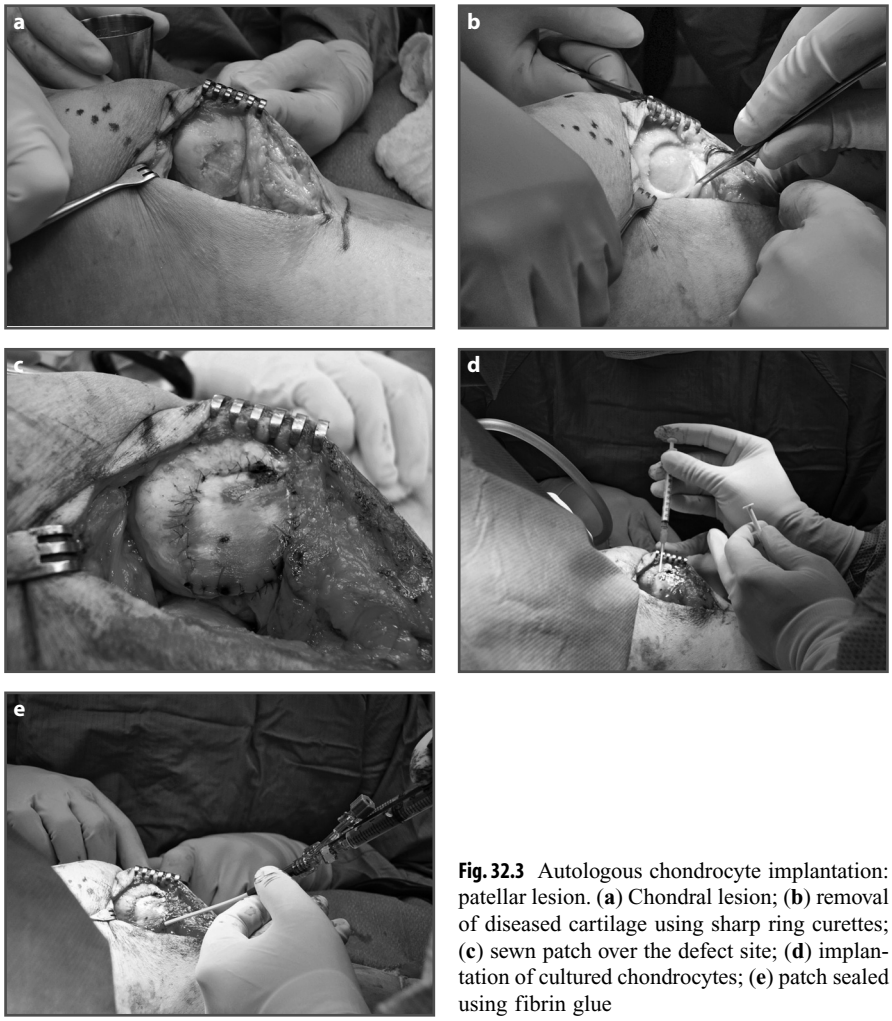

Fig. 32.3 Autologous chondrocyte implantation: patellar lesion. (**a**) Chondral lesion; (**b**) removal of diseased cartilage using sharp ring curettes; (**c**) sewn patch over the defect site; (**d**) implantation of cultured chondrocytes; (**e**) patch sealed using fibrin glue

Post-operatively, patients with a femoral condyle lesion are kept in non-weight-bearing or partial weight-bearing, with CPM rehabilitation for up to 6 weeks. Similarly, patients with a patellofemoral lesion are advised to use CPM for 6-8 h daily, but are permitted full weight-bearing with the knee in extension. A return to the normal activities of daily living and sport activities is allowed 6 months after surgery, as ACI results in a "hyaline-like" cartilage believed to be biomechanically superior to fibrocartilage. Table 32.3 summarizes the outcome studies for ACI.

Table 32.3 Clinical outcomes of autologous chondrocyte implantation (ACI)

Authors	Study group	Lesion characteristics and site	Outcomes	Comments
Mandelbaum et al. [23]	40 ACI Age 16-48 years Mean follow-up 59 months	Trochlear lesions Mean lesion size 4.5 cm^2	Modified Cincinnati knee improvement (3.1 to 6.4)[a] Pain score improvement (2.6 to 6.2) Swelling score improvement (3.9 to 6.3) No failed implants	
Zaslava et al. [24]	126 ACI Mean age 34.5 years Mean lesion size 4.63 cm^2 Mean follow-up 48 months	102 medial femoral condyle 27 lateral femoral condyle 24 trochlea	76% success VAS improvement (28.8 to 69.9) Modified Cincinnati knee improvement (3.3 to 6.3) SF-36 Improvement (33.0 to 44.4)	
Rosemberg et al. [25]	56 ACI Age 45-60 years Mean follow-up 4.7 years	Mean lesion size 4.7 cm^2	8 failures (14%) lasting improvement in 88% of patients Good or excellent at last follow-up self-rated by 72% of patients Modified Cincinnati knee improvement (3.6 to 5.9) SF-36 score improvement (31.6 to 45.6)	Failure rate of ACI in older patients comparable with rates reported in younger patient groups
Steinwachs and Kreutz [26]	63 ACI Mean age 34 years 36 months follow-up	34 femoral condyle 10 trochlea 19 patella Mean lesion size 5.85 cm^2	Significant improvement in ICRS and modified Cincinnati scores	Use of a collagen membrane avoids graft hypertrophy

[a] Significant $p < 0.05$

(*continued* →)

Table 32.3 (*continued* →)

Authors	Study group	Lesion characteristics and site	Outcomes	Comments
Kreuz et al. [27]	118 ACI Mean age 35 years (range 18-50) 36 months follow-up	78 femoral condyle 17 trochlea 23 patella Mean lesion size 6.5 cm^2	Improvement in Cincinnati and ICRS scores Improvement in MRI parameters (defect filling, subchondral edema, cartilage signal and effusion)	Patients involved in regular (1-3 times/week) or competitive (4-7 times/week) sport activities had significantly better outcome than patients with no or rare sports involvement
McNickle et al. [28]	137 patients (140 knees) Mean age 30.3 years Mean follow-up 4.3 years	24 LFC 62 MFC 41 patella 13 trochlea Mean lesion size 5.2 cm^2	Lysholm improvement (41 to 69)[a] IKDC scores Improvement (34 to 64)[a] Debridement of the autologous chondrocyte implantation site secondary to persistent symptoms in 21 patients (16%) Revision procedure in 9 knees (6.4%) Completely or mostly satisfied subjectively reported by 75% of patients	

[a] Significant $p < 0.05$

Osteochondral grafting involves implantation of a cylindrical plug of articular cartilage and subchondral bone at the defect site. The graft source can be from the host (autograft) or from a cadaveric donor (allograft).

Osteochondral autograft (OAT) is advantageous by virtue of using the patient's own tissue, eliminating immunologic concerns and the potential of disease transmission. The autograft is most commonly harvested through a small incision from a non-weight-bearing region of the knee, where the articular cartilage and the underlying bone can be removed without inducing new symptoms or loss of function. The harvested cylindrical plug is most often inserted arthroscopically at the lesion site using a press-fit technique. Nonetheless, autograft transplantation is greatly limited by the supply of cartilage available from a non-weight-bearing area and by donor site morbidity. In general, autografts are indicated in symptomatic patients with small full-thickness defects ($< 2\ cm^2$) and limited subchondral bone loss (< 6 mm), or as a revision procedure for

a previously failed microfracture or ACI. For larger lesions, a technique using multiple plugs called "mosaicplasty" can be employed; however, the corresponding author prefers to use a smaller number of larger-diameter plugs. Post-operatively, patients are protected from full weight-bearing for 4-6 weeks and are advised to use a CPM machine for 4-6 h each day. Surgical outcomes of the OAT procedure are presented in Table 32.3.

Osteochondral allograft (OA) involves the transplantation of mature hyaline cartilage with intact native architecture and living chondrocytes from a donor. OA grafts are indicated for larger defects (> 2.5 cm^2) or those with associated bone loss (avascular necrosis, osteochondral fractures, and osteochondritis dissecans). This procedure is most often used as a secondary treatment option in patients who have failed previous attempt at cartilage repair, but can be a first-line treatment for high-demand patients with large lesions [16]. Most commonly, OA grafts are used for medium-sized to large articular cartilage defects involving the femoral condyle, but may also be used for lesions of the tibial plateau, trochlea, and patella. Major concerns associated with allograft transplantation, such as tissue mismatching and immunologic response, do not play a major role in OA grafting, as the transplanted tissue is avascular and alymphatic. In addition, before transplantation, the donor tissue is washed using pulsatile irrigation in an attempt to remove marrow elements that may contribute to suboptimal graft incorporation. However, one of the challenges faced by the operating surgeon is the availability of allograft donor tissue. Clinical studies have suggested that there is higher chondrocyte viability and subsequently improved maintenance of cartilage matrix in fresh grafts, and most surgeons currently prefer the use of fresh OA grafts. Once harvested, the fresh grafts are kept in physiologic medium at 4°C to preserve chondrocyte viability. The storage time has also been shown to have an effect on chondrocyte viability, with fewer viable cells observed with prolonged storage [32]. Studies have shown a significant decrease in cell viability after 14 days, from 91.2% to 80.2%, with further detrimental effects at 28 days, with a cell viability of 28.9% [33]. The current recommendation is that a fresh OA graft should ideally be used by 14 days and certainly no longer than 28 days from harvest [34-36].

OA graft transplantation is typically performed through a small arthrotomy to expose the lesion site (Arthrex, Naples, FL). The preparation of the graft involves the use of a reamer to convert the defect into a circular recipient socket with a uniform depth of 6-8 mm. An instrumentation system is used to size and harvest a cylindrical plug from the allograft, which is then implanted into the socket after careful alignment of the four quadrants to the recipient site. If a large allograft is used, fixation may be augmented with a bioabsorbable or metal compression screw (Fig. 32.4).

Post-operative rehabilitation consists of immediate CPM and weight-bearing limited to toe-touch for 6 weeks. Patients with patellofemoral grafts are allowed to weight bear as tolerated in extension, with flexion generally limited to 45° for 4 weeks. A return to the normal activities of daily living and sport activity is considered at 8-12 months. Post-surgical outcomes of OA grafts are presented in Table 32.4.

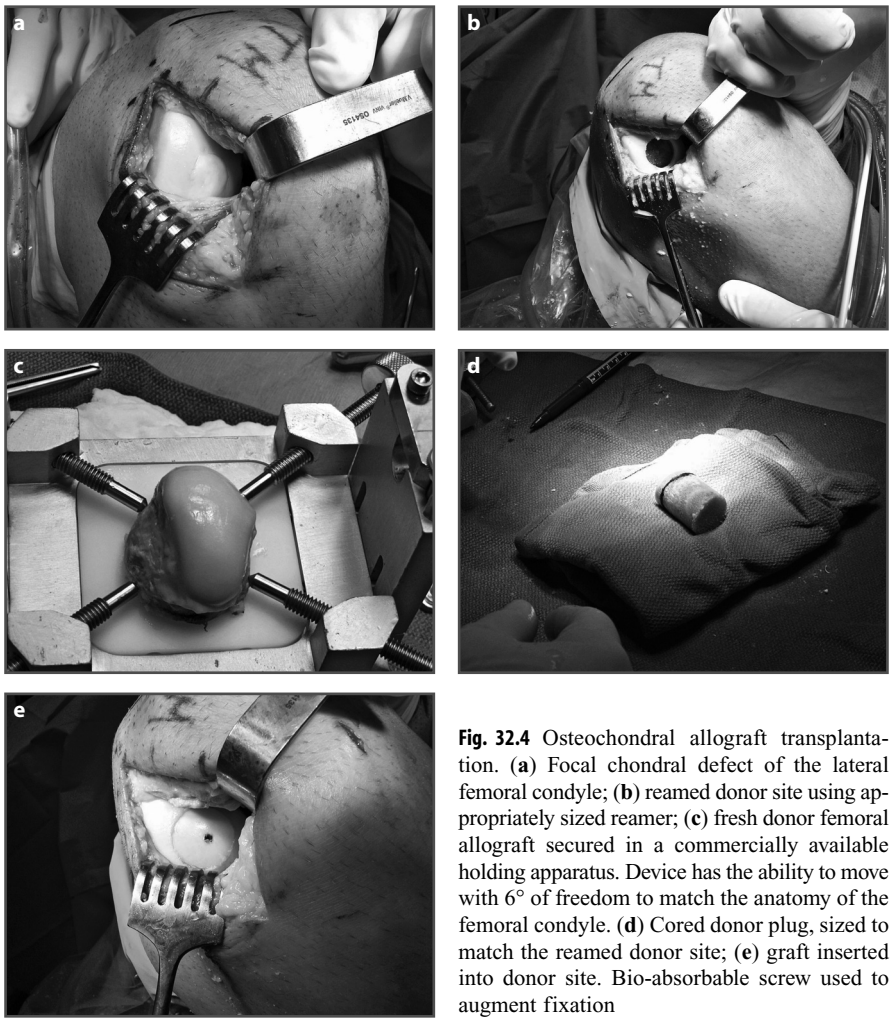

Fig. 32.4 Osteochondral allograft transplantation. (**a**) Focal chondral defect of the lateral femoral condyle; (**b**) reamed donor site using appropriately sized reamer; (**c**) fresh donor femoral allograft secured in a commercially available holding apparatus. Device has the ability to move with 6° of freedom to match the anatomy of the femoral condyle. (**d**) Cored donor plug, sized to match the reamed donor site; (**e**) graft inserted into donor site. Bio-absorbable screw used to augment fixation

32.6.3 Concomitant Procedures

Combined pathologies are frequently encountered by the operating surgeon treating articular cartilage defects. Meniscal injury or deficiency, malalignment, and ligamentous instability are known to contribute to the development of articular lesions. Surgically addressing these concomitant pathologies is crucial for an effective and durable cartilage repair, ensures the integrity of the primary cartilage repair, and does not negatively affect the patient's ability to return to his or her day-to-day activities. It is also advantageous to treat combined pathologies at the time of primary cartilage repair, thus sparing the patient a prolonged rehabilitation.

Table 32.4 Clinical outcomes of osteochondral autograft (OAT)

Authors	Study group	Lesion characteristics and site	Outcomes	Comments
Hangody et al. [29]	1097 cases Mean age 36 years Mean follow-up 14 years	798 femoral condyle 147 patello-femoral 31 tibia 120 others than knee Small and medium focal defects (1-4 cm^2)	Good/excellent results in 92% of femoral condylar implants, 87% of tibial resurfacements, 74% of patellar and/or trochlear mosaicplasties Graft survival in 81 of 98 cases	Histological findings: survival of the transplanted hyaline cartilage; fibrocartilage covering the donor sites
Marcacci et al. [30]	30 cases Mean age 29.3 years 2- and 7-year follow-up	MFC LFC Mean lesion size < 2.5 cm^2	76.7% report good/excellent results according to ICRS objective evaluation IKDC subjective score improvement (34.8 to 71.8) Tegner score improvement (2.9 to 5.6 at 7 years) MRI findings: good integration and survival of the graft in 62.5% of patients at 7 years	Reduced sports activity from 2- to 7 year follow-up
Dozin et al. [31]	25 patients Mean age 27 years Median follow-up 300 days	8% femoral condyles 32% patella Mean lesion size 1.88 cm^2	Improvement in Lysholm score in 88% of patients No failures	

32.7 Conclusion

Articular cartilage repair is aimed at returning patients back to their pre-injury level of activities. While nutritional supplements may play a role in the prevention and treatment of cartilage injuries, more often than not surgical intervention is required. Several surgical techniques have been shown to alleviate patient's symptoms and improve function but determining which technique to apply requires a thorough clinical history that includes the patient's age, activity level, and post-operative expectations, as well as the size of the lesion. Each technique is associated with specific advantages and limitations. Second-generation techniques are under development to improve current shortcomings.

Table 32.5 Clinical outcomes of osteochondral allograft graft

Authors	Study group	Lesion characteristics and site	Outcomes	Comments
Gross AE et al. [37]	60 fresh allografts Mean age 27 years Mean follow-up 10 years	30 MFC 30 LFC	12 failures Survival: 95% at 5 years, 85% at 10 years, 65% at 15 years	
Davidson et al. [38]	Fresh allografts in 8 patients (10 knees), Mean age 32.6 years Mean follow-up 40 months	6 MFC 2 MFC 2 trochlea Mean defect size 6.2 cm^2 (range 2.5-17.2 cm^2)	No failures Improvement in IKDC score, SF-36, Tegner, Lysholm	Histological findings: cellular density and viability similar in host and donor cartilage MRI: complete incorporation and improvement in scores
McCulloch et al. [39]	25 fresh allografts Mean age 35 years (range 17-49) Mean follow-up 35 months	Femoral condyle	Improvement in Lysholm, IKDC, KOOS, SF-12 scores 88% (22) of radiographs showed graft in corporation at last follow-up	
Jamali et al. [40]	Fresh allograft in 18 patients (20 knees) Mean age 42 years (range 19-64) Mean follow-up 94 months	Patellofemoral No re-alignment procedure performed	5 failures 60% (12 of 20) excellent/good results Improvement in clinical scores	Clinical and radiographic study

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