

## **CARTILAGE RESTORATION**

*BOOK TITLE: Knee Reconstruction, Replacement and Revision*

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### **Introduction**

Articular cartilage can experience both acute injury and chronic degeneration. These processes vary with patient age, pathologic insult, natural history, and future prognosis. There is often an overlap between the two; therefore, injury or degeneration of articular cartilage should be thought of as a spectrum rather than a discreet mechanism. However, regardless of the pathologic insult, articular cartilage has a limited capacity for repair. This is most likely due to a multitude of factors. Poor vascular supply requires interstitial fluid to provide the necessary nutrients via diffusion. To further complicate the repair process, chondrocytes have low mitotic activity and a low turnover rate. Lastly, cartilage has a deficiency in the capacity for an undifferentiated cell population to respond to the damage.

The natural history of isolated chondral and osteochondral defects is unknown. However, clinical experience suggests that, when left untreated, these lesions do not heal and may progress to symptomatic degeneration of the joint. Furthermore, lesion progression may be dependent on size, location, subchondral bone, age of patient, limb alignment, BMI and joint stability. The available natural history studies suffer from small patient populations with confounding co-existing pathology. To further complicate the matter, some studies have suggested that radiographic deterioration may be present, but this is unrelated to the functional scores<sup>1</sup>.

In our clinical experience, early surgical intervention for symptomatic lesions is often suggested in an effort to restore normal joint congruity and pressure distribution and prevent further injury. However, the surgeon must ensure that the expectations of the patient and the goals of surgical treatment are aligned. The most predictable goal with surgery is to provide pain relief and improve joint function, thus allowing patients to comfortably perform activities of daily living. These patients do also have the potential to attain or return to a higher level of sport/activity, but these outcomes are significantly more variable.

Multiple algorithms have been described in an effort to simplify the treatment of cartilage lesions. These provide an important dynamic conceptual framework that creates consistent management of patient pathology. However, these algorithms must be flexible as new concepts and information arise. In general, surgical options can be grouped into three categories: palliative (arthroscopic débridement and lavage), reparative (marrow stimulation techniques), and restorative (osteochondral grafting and autologous chondrocyte implantation). All of these

techniques have been reported to improve the clinical status as compared with the preoperative state. Thus, the appropriate treatment for any given cartilage lesion is patient-specific. Important considerations include; the size and location of the lesion, the physical demands of the patient, and the treatment history. A realistic and comprehensive understanding of the patient's goals is critical to any decision regarding how to treat a symptomatic chondral defect. In keeping with these principles, the treatment algorithm consists of a graduated surgical plan. The least destructive and least invasive treatment option necessary to alleviate the symptoms and restore joint function is performed first. The more extensive treatments are reserved for potential salvage operations later.

## **Preoperative Evaluation**

### ***History/Examination***

Patient specific factors are as important as defect characteristics when formulating a treatment plan. History, physical examination, and articular pathology must all correlate. An example of the importance of this would be a patient with a known classic osteochondritis dissecans of the medial femoral condyle who reports bilateral anterior knee pain with stair-climbing. The patient's history is consistent with patellofemoral pain and the evaluation should focus on this. The presence of a cartilage lesion may be a confounding factor that is not contributing to the discomfort.

In the evaluation of a patient with a suspected cartilage lesion there are many patient factors that must be taken into account. Patient age, body mass index, occupation and/or family commitments, risk-aversion (desire to avoid subsequent surgical procedures), responsiveness and rehabilitation after previous surgical treatments, and the patient's specific concerns related to his or her problem are all important preoperative considerations. As evidenced by the previous criteria, the approach to articular cartilage lesion must encompass a "total patient" view in addition to a focused extremity specific evaluation. With regard to the affected knee, the important history components include: weight-bearing vs non weight-bearing pain, swelling, mechanical symptoms, giving-way, and aggravation of symptoms related to walking on level ground as opposed to stair-climbing.

Physical examination should include a standard knee examination with special emphasis on point tenderness, location of pain, limb alignment and stability. These all provide vital information in the formulation of a treatment plan. A mal-aligned extremity must be corrected if a symptomatic cartilage lesion is to be successfully treated, and an unstable knee can complicate the results of any treatment.

Lastly, a discussion should be had in order to address and understand the patient's specific concerns and goals. This is essential to achieve a successful outcome given the complexity of the pathology and associated treatments. Some concerns that patients may have include whether it is safe to remain active despite symptoms and whether a delay in surgical intervention precludes certain treatment options because of disease progression. This is a difficult discussion due to our lack of understanding regarding the natural history of these defects

and therefore difficult to advise patients. It is best to carry out careful discussions on a case-by-case basis.

## **Imaging**

Radiographs are essential in the initial evaluation of articular cartilage disease. Bipolar degenerative changes can be evident with decreased joint space and the assessment of overall limb alignment are both important factors in developing a treatment plan as will be discussed in the treatment section. Important radiographic views that are often overlooked (but important) include the Rosenberg view, which is a weightbearing PA of the knees in 30 degrees of flexion, and a full length (hip to ankle) view. These will help fully evaluate alignment and condylar degenerative/injury.

However, a complete evaluation of a suspected articular cartilage injury/degeneration should include an MRI scan of the affected knee. Contemporary imaging techniques have both significantly increased the quality of studies as well as the complexity of obtaining the best images. Thus, in order to better understand the lesions we are treating, a discussion of MRI imaging techniques is warranted. They are important for not just the initial workup, but also for monitoring the status of any treatment rendered. MRI scans can be broken down into two basic categories; morphologic and composition analysis. Current MRI imaging techniques for morphologic analysis of cartilage, include; conventional spin-echo (SE) and gradient-recalled echo (GRE) sequences, fast SE sequences, and more advanced isotropic three-dimensional (3D) SE and GRE sequences. Compositional assessment techniques include T2 mapping, delayed gadolinium-enhanced MR imaging of cartilage (dGEMRIC), T1ρ imaging, sodium imaging, and diffusion-weighted imaging.

The sequences most commonly used in the assessment of joint cartilage are 2D or multisection T1-weighted, proton density-weighted, and T2-weighted imaging sequences with or without fat suppression. In general, T1-weighted images show intrasubstance anatomic detail of hyaline cartilage but do not provide good contrast at the cartilage surface. This disadvantage limits the usefulness for assessing focal cartilaginous defects. T2-weighted imaging provides good contrast between the cartilage surface and joint fluid, which is useful for detecting focal areas of delamination or other defects. However, it does not show the internal structure as well as T1. Proton density-weighted imaging is capable of showing both surface and internal characteristics, but there are nuances to its use that makes it not as clinically applicable as a combination of T1 and T2 imaging.

The future of cartilage assessment is moving towards an analysis of the intrasubstance content (Table 1). T2 mapping of hyaline articular cartilage reflects interactions among water molecules and between water molecules and surrounding macromolecules. It is very sensitive to changes in the internal matrix. Increased interactions between water and macromolecules (collagen) result in decreased T2. Thus, T2 is sensitive to changes in hydration and this can reflect the collagen concentration. The dGEMRIC technique is based on the observation that ions within the interstitial fluid of hyaline cartilage are distributed in relation to the concentration

of negatively charged glycosaminoglycan (GAG) molecules. Thus, when a negatively charged intravenous gadolinium substance (Gd-DTPA2) is administered it distributes based on the molecular content and concentrates in areas of low glycosaminoglycan content. This can then be visualized and quantified with MRI.

Sodium imaging is in the early stage development and validation. Sodium imaging is also based on the fact that healthy intracellular matrix has a negative charge and positive charges will be distributed respectively. Areas of high glycosaminoglycan content will have a large negative charge and sodium will then concentrate in these areas, whereas injured or degenerated cartilage will have a low glycosaminoglycan (negative) content. These sodium molecules can be imaged on specific MRI spin sequences. The advantage of sodium imaging is that it is a naturally occurring substance and does not require the administration and uptake of a contrast agent. Overall, MRI is an extremely powerful tool in the evaluation of cartilage structure and composition. As the availability increases and the efficacy of specific techniques is refined, we will most likely see changes in treatment based on the health of articular cartilage as well as an exponentially better ability to assess our treatment strategies.

Regardless of the technique utilized, the evaluation of a cartilage defect should include specific variables; defect location, number, size, depth, and geometry; the condition of the subchondral bone and surrounding cartilage; and the degree of containment. The condition of the apposing surface, which is often overlooked, is also an important variable. Even minor areas of early degeneration make achieving a satisfactory clinical outcome challenging. Specific management of each of these defect-specific variables increases the likelihood of a good clinical outcome.

Finally, one must carefully search for associated pathological conditions, such as malalignment, ligament insufficiency, and concomitant meniscal deficiency that may contribute to treatment failure and should be corrected before or during the surgery to treat the chondral lesion.

### **Treatment Algorithm**

The first step in determining the appropriate treatment algorithm is to assess for any associated pathology that may need to be addressed. Malalignment, ligament insufficiency, and meniscal deficiency can contribute to the clinical manifestation of a lesion on the femoral condyle and should be treated with either a staged or concomitant procedure. In some cases, a high tibial osteotomy or ligament reconstruction may prove to be the definitive procedure. In other cases, these procedures may be “protective” of the cartilage reconstruction. Research has shown that patients with malalignment will have worse clinical outcomes than those with appropriate alignment. With regard to patellofemoral lesions, these are often treated with a simultaneous realignment procedure (anteromedialization of the tibial tuberosity). However, anteromedialization is more successful for lateral patellofemoral lesions than it is for lesions located along the medial aspect of the patellofemoral joint. Medial patellofemoral lesions can be treated with a more vertically oriented anteromedialization. Regardless, in order to provide the

patient with the best possible outcome, all associated pathology should be fully evaluated and addressed when necessary.

The treatment algorithm for specific chondral lesions is guided by the lesion size/location and the patient activity level (Figures 1 and 2). These treatments can then be broken down into palliative, reparative, and regenerative categories. Palliative treatments would include conservative management and debridement techniques. In some patients with diffuse disease and mechanical symptoms debridement may be effective, but for the large majority of discrete lesions this does not provide much relief. In these cases, the first line treatment is reparative with marrow stimulation techniques. Microfracture is often used for smaller lesions ( $<2 \text{ cm}^2$ ), or in patients with larger lesions ( $>3 \text{ cm}^2$ ) and modest physical or physiologic demand levels. However, small lesions in high-demand patients may require regenerative techniques including osteochondral autografts. Larger lesions ( $>2.5 \text{ cm}^2$ ) are typically more amenable to osteochondral allografting or autologous chondrocyte implantation. Autologous chondrocyte implantation is advised for younger patients with shallow lesions, especially of the patellofemoral joint. This method does not violate the subchondral bone and minimizes the impact on future treatment such as osteochondral allograft transplantation. Larger, deeper lesions with bone loss typically require an osteochondral allograft. More recently, a minimally manipulated tissue alternative using allogeneic juvenile particulate articular cartilage (DeNovo NT, Zimmer, Inc., Warsaw, IA) has demonstrated early favorable results for the treatment of symptomatic chondral defects of the knee.

Treatment is also guided by the location of the lesion. For example, osteochondral allografts are used for femoral condyle lesions because they allow accurate anatomic reconstruction. Whereas, lesions of the patellofemoral joint are often treated with autologous chondrocyte implantation because they are small and the varying anatomic concavity and convexity make structural grafts too difficult to fit in place. The tibia remains a difficult articular surface to treat and small tibial lesions can be treated with marrow stimulation techniques. Other options include the utilization of osteochondral autografts placed in a retrograde manner with use of a cannulated reamer system (Arthrex, Naples, Florida). The use of osteochondral allografts with an intact meniscus and concomitant realignment has been reported for the treatment of larger lesions of the tibial plateau, especially after fracture and the development of secondary arthritis, with graft survival rates of up to 65% at fifteen years<sup>2</sup>.

## **Techniques**

### **Marrow Stimulation Technique (Microfracture)**

The microfracture marrow stimulation technique is carried out with a surgical awl to penetrate the subchondral bone. This promotes bleeding and the surfacing of local stem cells and other anabolic factors that support the formation of a clot. This clot, along with the pluripotent stem cells, serves as the foundation for a reparative fibrocartilage tissue.

Critical to the success of this technique is the creation of vertical walls and a stable articular cartilage lesion. This improves the local mechanical environment during healing by

reducing shear and compression. All unstable cartilage is removed when the lesion site is prepared. The calcified cartilage layer is carefully débrided, and surgical awls are used to penetrate the subchondral bone (Figure 3). The holes are placed perpendicular to the bone surface, 2 to 3 mm apart.

### ***Rehabilitation***

Postoperative rehabilitation is guided by the location of the lesion, but typically it involves up to six weeks of non-weight-bearing and the use of a continuous-passive-motion machine for six hours per day if the cost is not prohibitive. Patients with a lesion in the patellofemoral joint wear a brace with a flexion stop of 30° to limit patellofemoral contact; weight-bearing is permitted.

### ***Outcomes***

The best outcomes of this technique are seen in younger patients with small traumatic lesions. After two and five years of follow-up, Knutsen et al.<sup>3</sup> found no difference between the outcomes of microfracture and those of autologous chondrocyte implantation for femoral condyle lesions, but patients with smaller lesions treated with microfracture did better than those with larger lesions. Similarly, Gudas et al. observed that, among patients with lesions exceeding 2 cm<sup>2</sup> in the central part of the medial femoral condyle, those treated with microfracture had lower clinical outcome scores than did those treated with an osteochondral autograft transplantation.<sup>4</sup> Location also plays a role in the success of marrow stimulation techniques, with better results seen after the treatment of femoral condyle lesions.<sup>5</sup> Lastly, Steadman et al<sup>6</sup> has shown good long term results with microfracture alone for traumatic defects.

### **Osteochondral Autograft Transplantation (OATS)**

Osteochondral autograft transplantation and mosaicplasty are the transfer of one or more cylindrical osteochondral autografts into a cartilage defect. The autografts are harvested from the non-weight-bearing periphery of the femoral trochlea or the margin of the intercondylar notch. With a combination of different graft sizes, 90% to 100% of the defect can be filled. However, this technique is limited by the amount of donor tissue available in the knee, and donor site morbidity increases as more tissue is harvested. Osteochondral autograft transplantation is best for small lesions (<2 cm<sup>2</sup>), but good clinical results have been reported with lesions between 2 and 4 cm<sup>2</sup>.<sup>7</sup>

Osteochondral autograft transplantation can be done through a small arthrotomy or entirely arthroscopically. To harvest donor grafts perpendicular to the surface, we prefer to obtain the donor plugs through a small lateral arthrotomy because the lateral edge of the patella can interfere with an arthroscopic harvest (Figure 4). The plugs are then implanted arthroscopically. There are many available commercial systems that provide a series of donor and recipient harvesting tubes to create a press-fit implant of up to 10 mm in diameter. A sizing guide is used to determine the number and size of grafts that are needed. A properly sized graft harvester with a collared pin is introduced perpendicular to the donor site (Figure 5) to a depth of

approximately 12 to 15 mm. The recipient socket is created to a depth that is 2 mm less than the length of the donor graft. It is important to maintain a perpendicular relationship between the donor graft and the articular surface to create well-defined vertical walls in the recipient socket, as this facilitates congruent plug placement. The donor plug is placed over the recipient site and gently advanced into the defect, where it is often left slightly proud. The chondrocytes can be damaged during impaction; therefore, it is critical to avoid high loads when inserting the graft. The final plug position should be flush with the surrounding articular cartilage (Figure 4).

### ***Rehabilitation***

Postoperatively, patients are protected from weight-bearing for six weeks and use a continuous-passive-motion machine six hours per day.

### ***Outcomes***

Hangody and Kárpáti evaluated the survival of the transplanted hyaline cartilage<sup>7</sup>. The graft undergoes osseous incorporation to the subchondral bone while the transplanted cartilage integrates with the adjacent host articular cartilage with fibrocartilage. Recently, Hangody et al. evaluated clinical outcomes at a mean of fourteen years after 1097 osteochondral autograft transplantation procedures as well as in an athletic population<sup>7,8</sup>. Encouraging results in this large multicenter series support the use of this technique for the treatment of small and medium focal chondral and osteochondral defects of the knee.

### **Osteochondral Allograft Transplantation**

Osteochondral allograft transplantation provides an option for treatment of larger lesions (>2.5 cm<sup>2</sup>) or those with substantial bone loss. It is normally a second-line treatment option, but can be a first-line treatment for high-demand patients with large lesions.

Osteochondral allograft transplantation can be used to resurface large, deep defects with mature hyaline articular cartilage while also filling any underlying osseous defect. Tissue matching and immunosuppression are not necessary because the transplanted chondrocytes are isolated by the cartilage matrix and not exposed to the host immune surveillance. The allografts can be “fresh” or frozen and can be implanted either open or arthroscopically. In the majority of cases we use a small arthrotomy to complete the procedure. The allograft is slowly warmed from 4°C to 37°C by placing it in normal saline solution at room temperature. The slow warming minimizes damage to the graft. The lesion is sized with a template, and a correspondingly sized reamer is used to convert the defect to a circular recipient socket with a uniform depth of 6 to 8 mm (Figure 6). This bone depth facilitates graft implantation and limits the amount of immunogenic donor bone that is implanted. A sterile marking pen is used to mark the 12 o'clock position of the lesion to orient the donor plug appropriately. An instrumentation system is used to size and harvest a cylindrical plug from the allograft (Figure 6). The donor graft is drilled through its entire depth with a harvester under irrigation with normal saline solution. The graft is extracted, and a ruler is used to measure and mark the four quadrants of the graft at the depth of the previously measured recipient sites. Before insertion, pulsatile lavage is used to remove the

residual blood and bone-marrow elements from the allograft to reduce the risk of disease transmission and graft immunogenicity. The graft is then press-fit into the socket by hand after careful alignment of the four quadrants to the recipient site (Figure 6). If the implanted allograft is particularly large, fixation may be augmented with bioabsorbable (Arthrex, Inc., Naples, FL) or metal compression screws.

### ***Rehabilitation***

Postoperatively, weight-bearing is limited to toe-touch for the first six weeks. Patients with a patellofemoral graft are allowed to bear weight as tolerated in extension and generally are limited to 45° of flexion during the first four weeks. Continuous passive motion is used immediately after the surgery. A return to normal activities of daily living and light sports activity is considered at eight to twelve months.

### ***Outcomes***

Subjective improvement can be expected in 75% to 85% of patients after osteochondral allograft implantation for properly selected chondral lesions. Multiple studies have supported this fact. Chu et al.<sup>9</sup> reported on fifty-five knees at a mean of six years after transplantation of fresh osteochondral allografts. Eighty-four percent of the knees treated for an isolated focal defect were rated as having a good-to-excellent outcome. However, only 50% of the knees that had undergone transplantation for the treatment of bipolar lesions had a good outcome. Ghazavi et al.<sup>10</sup> reported the results at a mean of 7.5 years following 126 procedures for the transplantation of fresh osteochondral allografts for the treatment of posttraumatic condylar defects. While a good-to-excellent result was achieved in 85% of the knees, an increased rate of failure was seen in the setting of bipolar lesions and limb malalignment. Furthermore, Davidson et al<sup>11</sup> showed an MRI improvement in outerbridge score with histologic analysis showing viability in host and donor tissue. McCulloch et al<sup>12</sup> also showed good results with 84% of patients satisfied and an 88% incorporation rate. Of note, again, patients with uncorrected alignment had worse results.

Unlike the femoral condyle, the patellofemoral joint does not have as good results. Jamali et al.<sup>13</sup>, reported only a 60% rate of good-to-excellent results at a mean of 7.8 years after transplantation of a fresh osteochondral allograft into the patellofemoral joint of twenty knees. Five knees in that series subsequently required salvage with revision allograft transplantation, patellectomy, arthrodesis, or total knee replacement

### **Autologous Chondrocyte Implantation**

Autologous chondrocyte implantation (ACI) is a good option for large contained defects from 2 to 10 cm<sup>2</sup> with bone loss of less than 6 to 8 mm. It is also a very good procedure for the patellofemoral joint with varying topography. ACI is typically a second-line treatment after a previous arthroscopic débridement has been performed. The first stage of autologous chondrocyte implantation is an arthroscopic evaluation of the size and depth of the focal chondral lesion and a cartilage biopsy. The total volume of the biopsied material should be



approximately 200 to 300 mg. The second stage is implantation of the cells (Figure 7). This is done usually no sooner than six weeks after the biopsy.

During implantation of the cultured cells, the defect is prepared by removing any existing fibrocartilage down to the underlying calcified layer. Vertical walls are created at the periphery of the lesion and complete hemostasis should be obtained with the tourniquet deflated. A patch is then sewn over the defect. Previously, we used a periosteal patch, however we are currently utilizing a synthetic collagen-membrane that is commercially available. These synthetic patches improve efficiency and avoid overgrowth associated with periosteum. Sutures (6-0 Vicryl [polyglactin]) are first passed into the patch approximately 2 mm from the edge and then passed through the cartilage at a depth of 2 to 3 mm below the cartilage surface. Sutures should be placed approximately 4 mm apart, and a gap should be maintained in the upper edge to allow chondrocyte implantation. The edges of the patch are sealed with fibrin glue, and a water-tightness test is performed with an 18-gauge angiocatheter. The chondrocytes are then delivered through the opening with use of an angiocatheter. After the cells have been implanted, the opening gap is closed with suture and fibrin glue.

### ***Rehabilitation***

Postoperatively, patients with a femoral condyle lesion are kept non-weight-bearing and use a continuous-passive-motion machine. Patients with a patellofemoral lesion are permitted full weight-bearing with the knee in extension. Continuous passive motion for six to eight hours per day at one cycle per minute is used for six weeks after the surgery. A return to normal activities of daily living and sports activities is allowed six months after the surgery.

### ***Results***

It is estimated that autologous chondrocyte implantation has been performed in >10,000 patients worldwide. The procedure has better results when it is done for lesions in the femoral condyle or in patients with a patellofemoral lesion who are undergoing a concomitant realignment procedure. There have been several studies comparing autologous chondrocyte implantation with other biologic reconstructive procedure. Peterson et al<sup>14</sup> showed good to excellent results in 51 of 61 patients at a mean follow-up of 7.4 years. Fu et al<sup>15</sup> described the results in 54 ACI patients and 42 debridement patients. At 3 years the ACI patients had lower pain and swelling scores than the debridement patients. Bentley et al<sup>16</sup> performed a level I prospective randomized clinical trial in which patients were randomized to either ACI or mosaicplasty (58 versus 42 patients, respectively). No significant difference was demonstrated in outcomes, and good to excellent results were obtained in 88% of patients treated with ACI and in 69% treated with mosaicplasty ( $P = 0.277$ ). Arthroscopy at 1 year demonstrated excellent or good repair tissue in 82% of patients after ACI versus in 34% of patients after mosaicplasty, based on ICRS grade. Knutsen et al<sup>3</sup> performed a randomized controlled study comparing ACI with microfracture. Eighty patients either received ACI or microfracture. At 12 and 24 months a significant improvement was noted in both groups. At 2-year follow-up, no statistically

significant difference was noted between the treatment groups. However, the microfracture group improved more than the ACI group based on the SF-36 physical component. A subsequent study evaluating the same trial patients at 5-year follow-up noted satisfactory results in 77% and no significant differences between the two treatment groups in any of the outcome measures. However, in a systematic review of ACI, it was found in 3 of 7 studies ACI had superior results to microfracture, 3 of 7 had no difference, and the one above had superior results with microfracture. Furthermore, microfracture results were found to deteriorate at 18-24 months and there is some histologic evidence that ACI or OATS produces lasting viable cartilage.<sup>17</sup>

### **Future Directions**

In the next 5-10 years the field of cartilage replacement will likely see the growth of single-stage cell based therapies. These are already in clinical use (CAIS, De Novo, AMIC, MACT) and as data accumulates, we will learn the efficacy and the appropriate patient populations for each technique<sup>18</sup>. Degenerative joint disease and traumatic articular cartilage lesions in the young patient remain a very difficult clinical entity to treat.

### **Case Examples**

#### ***Case #1***

The patient is a 21 year old female with right knee anterior pain and swelling for the previous 3 years. She has previously undergone a medial patellofemoral ligament reconstruction and an anteriomedialization for malalignment as well as a debridement. After the first procedure she did well for a year or two, then the pain recurred with activities of daily living. At the debridement procedure she was noted to have an 18 x 18 defect in her patella. Based on the fact that she has failed a realignment procedure and extensive conservative management, she was indicated for an ACI (Figures 8 and 9).

#### ***Case #2***

18 year old active male with 5 year history of activity related knee pain. Recently, pain has gotten worse and swelling has increased. He has undergone physical therapy from his primary doctor and been taking anti-inflammatories. The pain is bothering him on a daily basis and is localized to his medial joint line. Radiographs and arthroscopic views (Figure 10) show significant collapse and loss of his articular surface on the medial condyle. Treatment was an osteochondral allograft (Figure 11) and he is doing well 2 years post-op with minimal swelling and pain.

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## **Tables and Figures**

### ***Tables***

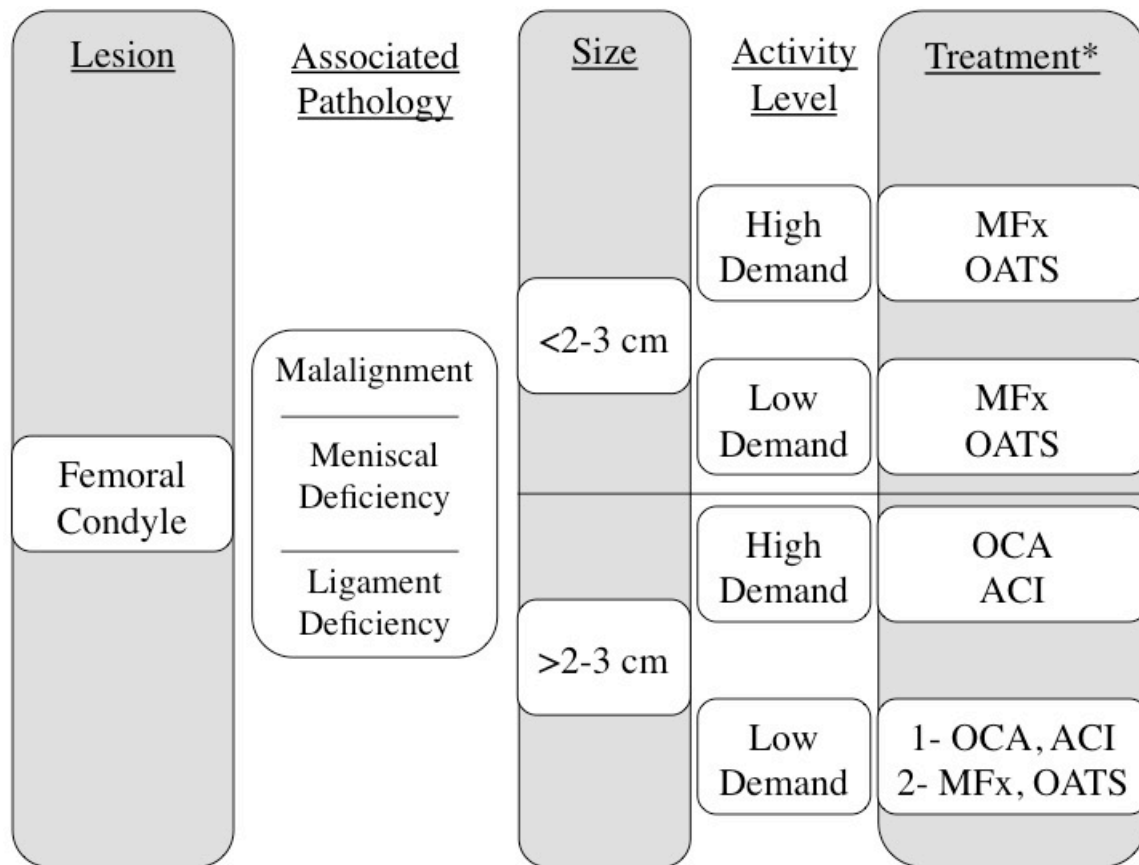
**Table 1** – Characteristics of MRI Techniques for the Assessment of Articular Cartilage Structure and Composition<sup>20</sup>

<b>MRI Technique</b>	<b>Intrasubstance Evaluation</b>	<b>Advantages</b>	<b>Disadvantages</b>
<b>T2 Mapping</b>	<ul style="list-style-type: none"> <li>• Collagen Structure</li> <li>• Water Content</li> </ul>	<ul style="list-style-type: none"> <li>• Clinically applicable</li> <li>• Validated in literature</li> <li>• No contrast needed</li> </ul>	<ul style="list-style-type: none"> <li>• Long acquisition time</li> </ul>
<b>dGEMRIC</b>	<ul style="list-style-type: none"> <li>• Glycosaminoglycans</li> </ul>	<ul style="list-style-type: none"> <li>• Measures correlates of GAG content</li> <li>• Validated</li> <li>• Clinically available</li> </ul>	<ul style="list-style-type: none"> <li>• IV contrast with time delay</li> </ul>
<b>T1ρ</b>	<ul style="list-style-type: none"> <li>• Collagen structure</li> </ul>	<ul style="list-style-type: none"> <li>• High sensitivity for early</li> </ul>	<ul style="list-style-type: none"> <li>• Long acquisition</li> </ul>

	<ul style="list-style-type: none"> <li>• Glycosaminoglycans</li> </ul>	<ul style="list-style-type: none"> <li>• degeneration</li> <li>• No contrast needed</li> </ul>	<ul style="list-style-type: none"> <li>• Specificity for cartilage component assessment needs further validation</li> <li>• Not tested in clinical trial</li> </ul>
<b>Sodium Imaging</b>	<ul style="list-style-type: none"> <li>• Glycosaminoglycans</li> </ul>	<ul style="list-style-type: none"> <li>• Directly measures GAG content</li> <li>• No contrast</li> </ul>	<ul style="list-style-type: none"> <li>• Low spatial resolution</li> <li>• Requires special hardware</li> <li>• Not tested in clinical trial</li> </ul>
<b>Diffusion Weighted Imaging</b>	<ul style="list-style-type: none"> <li>• Collagen structure</li> <li>• Glycosaminoglycans</li> </ul>	<ul style="list-style-type: none"> <li>• Provides info on GAGs in addition to previous techniques</li> <li>• No contrast</li> </ul>	<ul style="list-style-type: none"> <li>• Quantification to layers of cartilage is difficult</li> <li>• Not tested in clinical trial</li> </ul>

**Figures**

**Figure 1** – Flowchart for the management of femoral condyle defects. Treatment strategies are segmented in 1 = best and 2 = good treatments. MFx – Microfracture, OATS – Osteochondral Autograft Transplant, ACI – Autologous Chondrocyte Implantation, OCA – Osteochondral Allograft. \*DeNovo NT is an emerging option in the treatment of symptomatic articular cartilage defects.

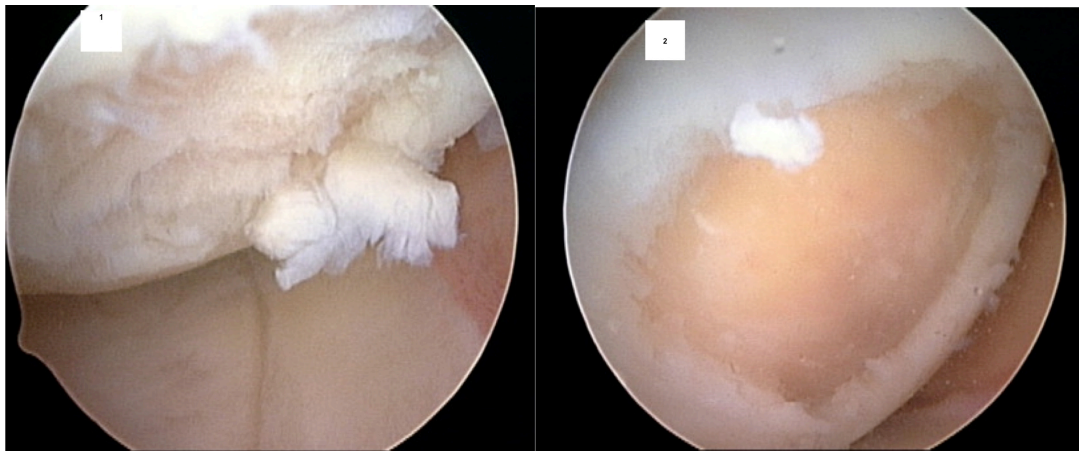


**Figure 2** – Flowchart for the treatment of patellofemoral defects. It should be noted that all patellofemoral pathology should initially be treated with significant rehabilitation and therapy.

Treatment strategies are segmented in 1 = best and 2 = good options. MFx – Microfracture, OATS – Osteochondral Autograft Transplant, ACI – Autologous Chondrocyte Implantation, OCA – Osteochondral Allograft, AMZ – Anteromedialization Procedure. \*DeNovo NT is an emerging option in the treatment of symptomatic articular cartilage defects.

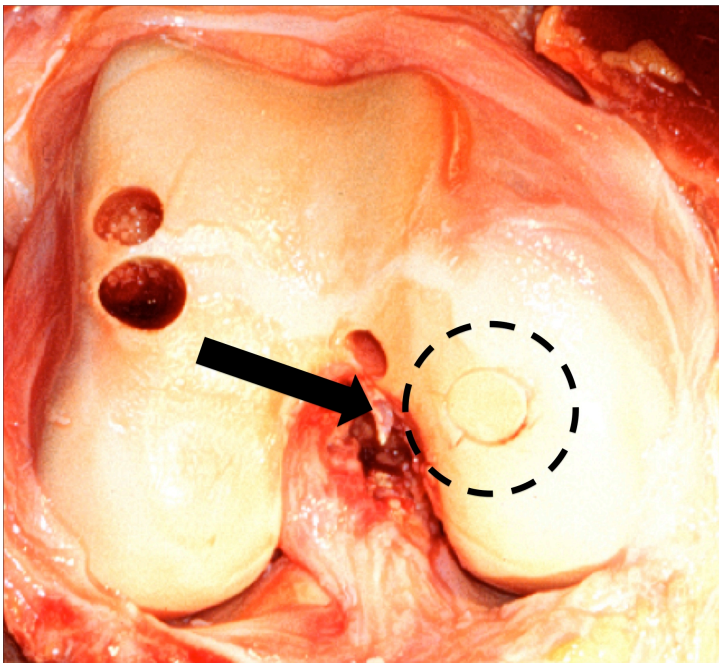
<u>Lesion</u>	<u>Associated Pathology</u>	<u>Size</u>	<u>Activity Level</u>	<u>Treatment*</u>
Patello-Femoral Joint	Patellofemoral Alignment	<2-3 cm	High Demand	1- ACI/AMZ 2- OATS/AMZ 2- OCA/AMZ
			Low Demand	1- MFx 2- ACI/AMZ
		>2-3 cm	High Demand	1- ACI/AMZ 1- OCA/AMZ
			Low Demand	1- MFx 2- ACI/AMZ

**Figure 3 - Microfracture.** 1 - A chondral lesion in the femoral condyle. 2 - The lesion was debrided with stable vertical borders. 3 - Microfracture holes were created in the subchondral bone 2 to 3 mm apart, beginning at the periphery of the lesion.



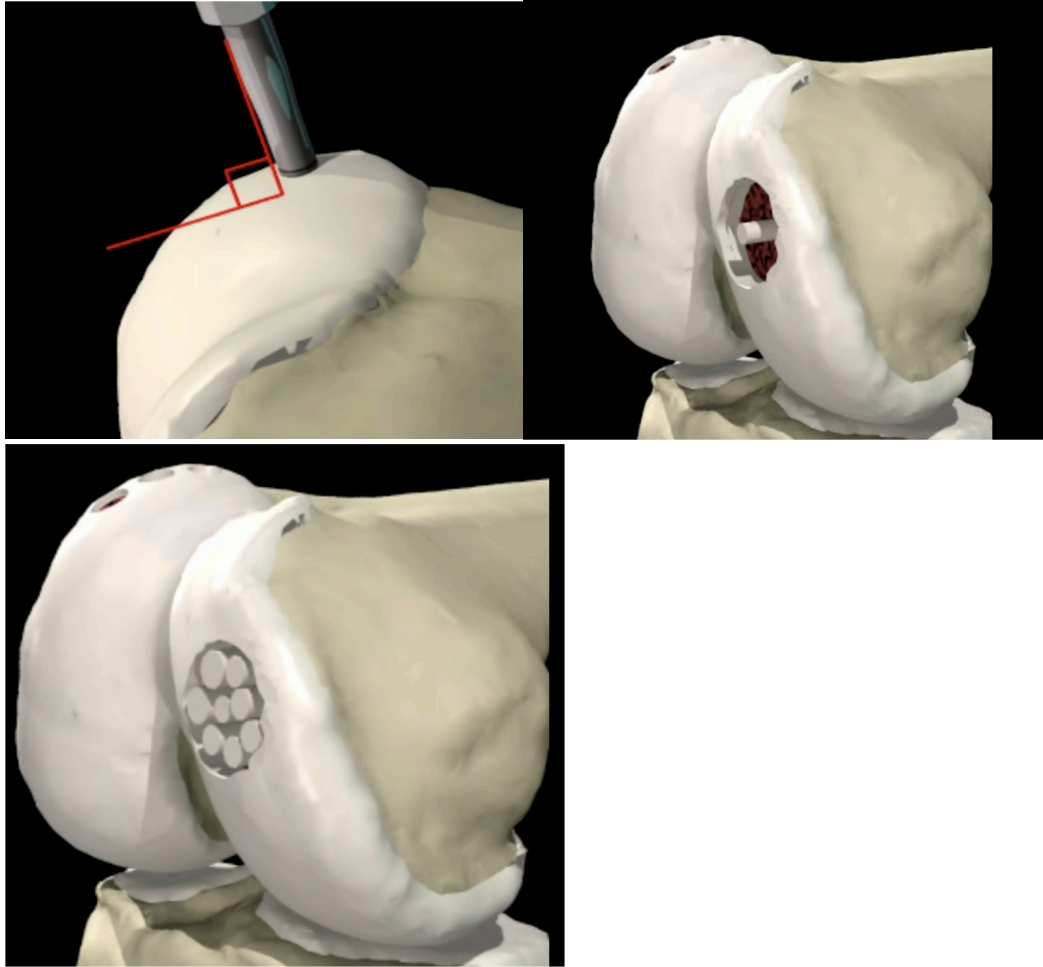


**Figure 4** – Osteochondral Autograft Transfer (OATS) – Cartilage from the non-articulating surface of the lateral condyle is moved to the chondral lesion.



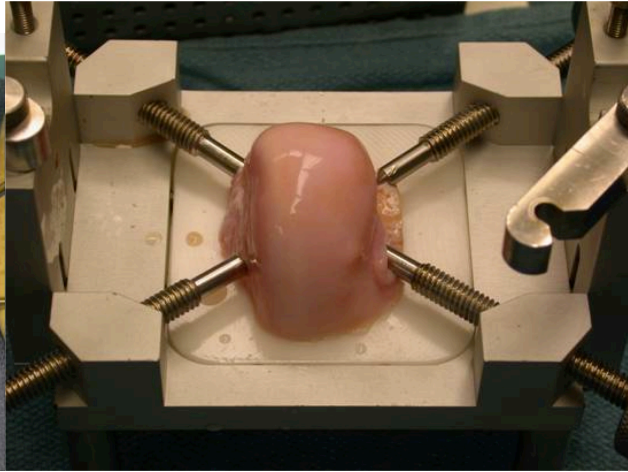
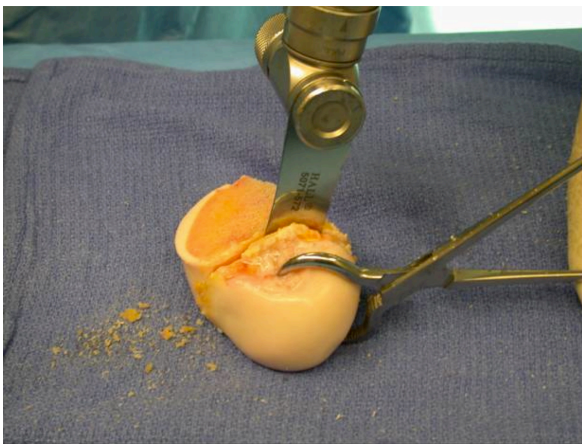
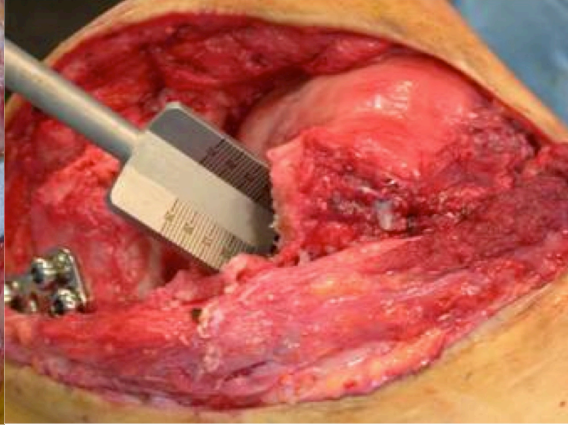
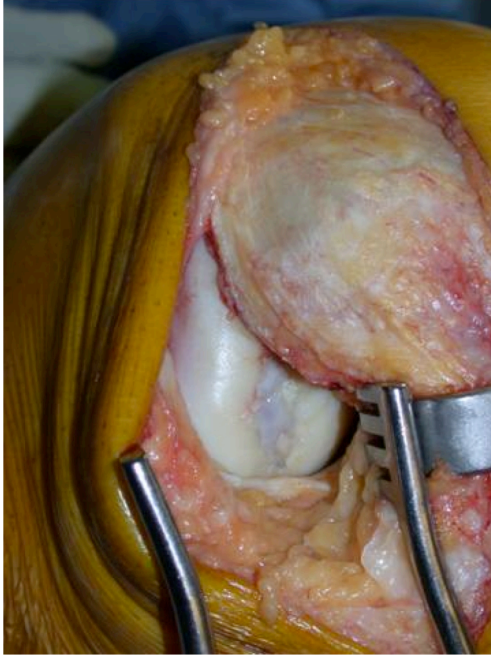
**Figure 5** – Mosaicplasty – Example of a generic commercially available system to harvest chondral plugs and place them into the defect. (Courtesy of Primal Pictures©)

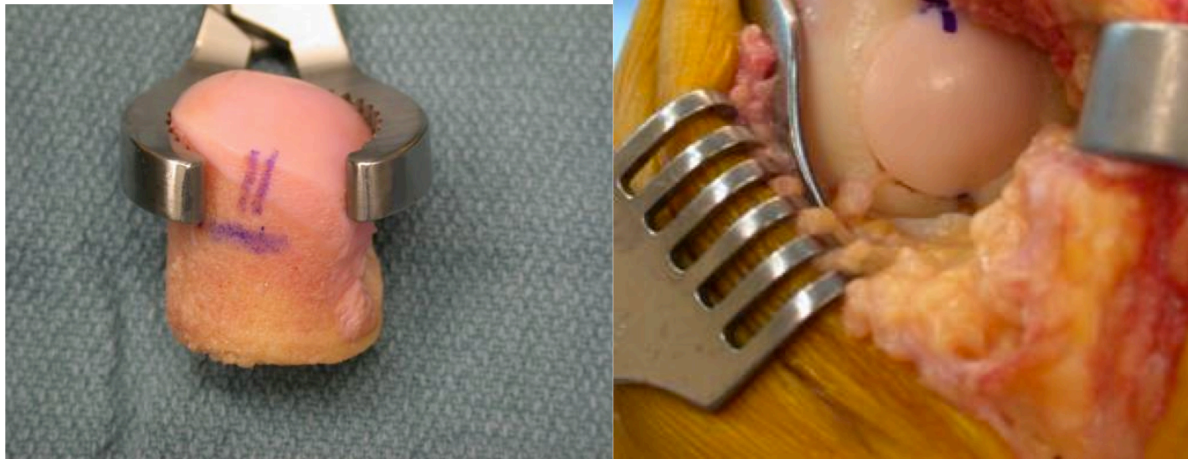




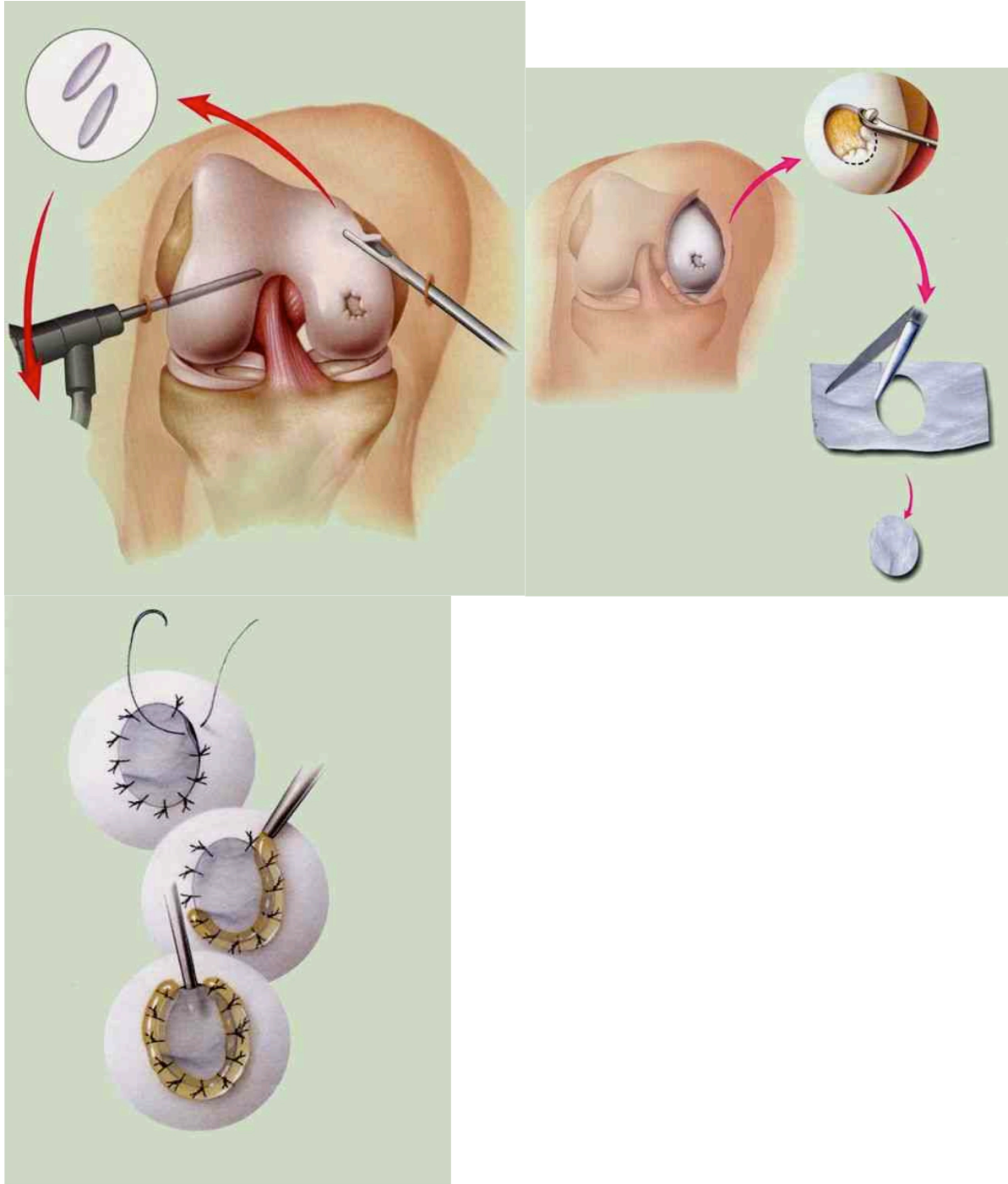
**Figure 6 – Osteochondral Allograft Technique -** 1: The procedure is typically performed through a small arthrotomy to expose the lesion. 2: A reamer is used to convert the defect to a circular recipient socket with a uniform depth of 6 to 8 mm. 3: Fresh donor femoral condyle. 4: The condyle is trimmed to create a flat surface to place on the workstation. This cut is made parallel to the potential harvest site. 5: Using a guide system, the graft is harvested from the condyle. 6: The graft is press-fit into the socket by hand after careful alignment of the four quadrants to the recipient site. The graft is flush with the recipient articular surface.







**Figure 7 – ACI** - 1: Cells are harvested at the first stage of the procedure. 2: The lesion is debrided and vertical walls are created. The lesion is sized and a synthetic patch is cut to the same specifications. 3: The patch is sewn into place and the cells are injected into the defect. Finally, fibrin glue is used to seal the construct. (Courtesy of Primal Pictures©)

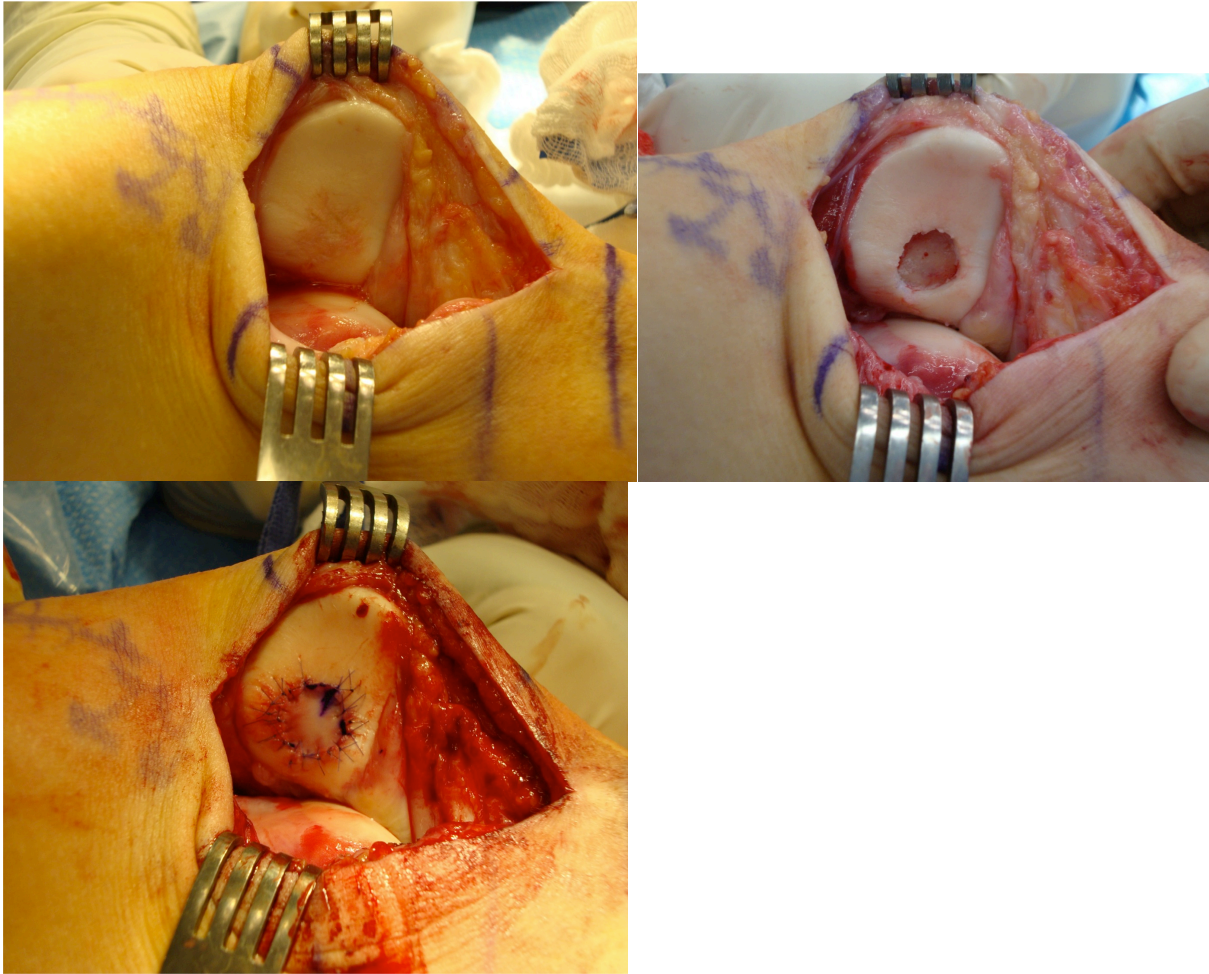


**Figure 8** – Radiographs of the patient with a patellar defect.

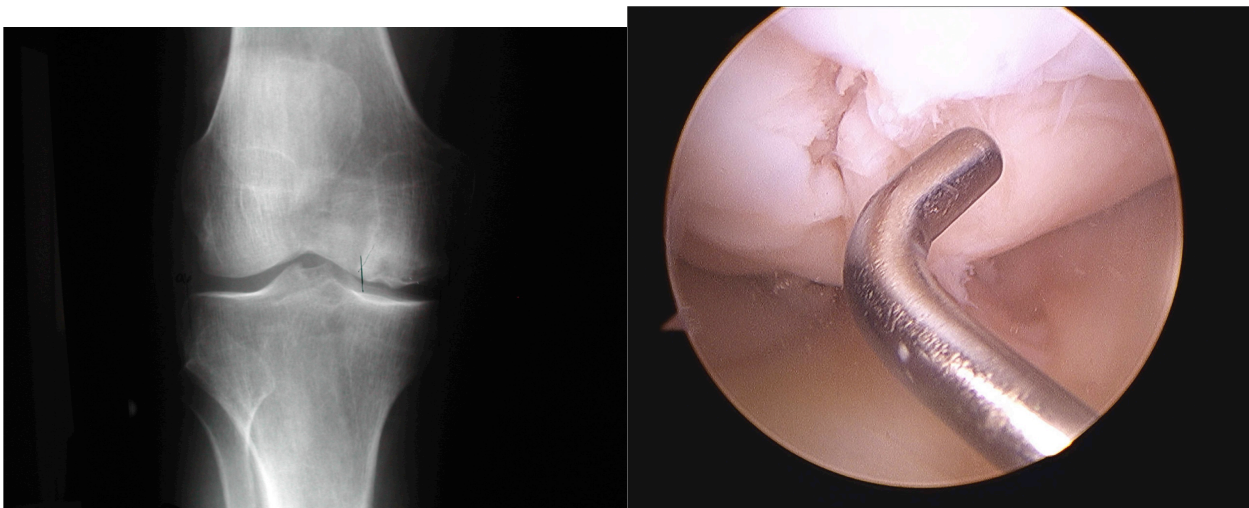


**Figure 9** - Autologous chondrocyte implantation - 1: A chondral lesion in the patella. 2: Preparation of the defect. 3: After the chondrocytes are delivered, the gap is closed with suture and fibrin glue.





**Figure 10** – Radiograph and arthroscopic picture of large medial femoral condyle chondral lesion.



**Figure 11** – 1: Medial femoral condyle articular defect. 2: After implantation of an

osteochondral allograft.

