CE

Innovations

Treatment Options for Articular Cartilage Defects of the Knee

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The treatment of symptomatic articular cartilage defects of the knee has evolved tremendously in the past decade. Previously, there were limited treatment options available to patients who suffered from either partial-thickness or full-thickness cartilage lesions. Because articular cartilage has a limited capacity for healing, patients were often treated symptomatically until they became candidates for osteotomy or total joint replacement. Recently, both reparative and restorative procedures have been developed to address this significant source of morbidity in young active patients. Microfracture is a reparative technique that induces a healing response to occur in an area of articular cartilage damage. Osteochondral autografts and allografts in addition to autologous chondrocyte implantation are restorative techniques aimed at recreating a more normal articular surface. Both types of procedures have been developed to alleviate the symptoms associated with focal chondral defects, as well as limit their potential to progress to a diffuse degenerative arthritis. Treatment can vary depending on both cartilage defect and patient factors. This article summarizes the various treatment options that have recently become available.

When articular cartilage is traumatically injured or has undergone degenerative changes resulting from arthritis, it is unable to heal the damaged cartilage with normal articular cartilage. Its lack of vascularity and relative absence of cells capable of becoming mature cartilage cells, or chondrocytes, make partial-thickness cartilage injuries incapable of healing or forming a new, smooth articular surface. Thus, partial-thickness cartilage injuries, without surgical intervention will either remain injured or progressively worsen with time. Pieces of articular cartilage may become elevated flaps and irritate the synovial lining of the knee causing swelling (effusion) and mechanical symptoms of catching. If the flap of cartilage becomes detached, it can become a loose body, causing locking of the knee so that it does not bend or straighten fully. Full-thickness cartilage injuries that also penetrate the subchondral bone are capable of limited healing with fibrocartilage, a type of cartilage that is different from normal articular cartilage and does not function as well or have the durability of normal cartilage. Patients with symptomatic cartilage defects previously were treated with anti-inflammatory medications, intraarticular steroid injections, intraarticular viscosupplements (hyaluronic acid), nutraceuticals (glucosamine or chondroitin sulfate), physical therapy, or activity modifications to alleviate their symptoms. Unfortunately, none of these treatment modalities results in cartilage healing. They may only decrease the associated pain or swelling. When quality of life is diminished despite the above treatments, osteotomies or total knee replacements were historically the major surgical options, but neither of these facilitated cartilage healing.

Over the past decade, surgical procedures have been developed to directly treat the cartilage injury by either reparative or restorative measures. Reparative procedures are designed to allow the cartilage lesion to heal with a different type of cartilage called fibrocartilage. This type of cartilage does not have the same mechanical properties as normal articular cartilage, hyaline cartilage, but does provide a covering over the otherwise exposed underlying bone, which can alleviate symptoms of pain and swelling. Restorative procedures are designed to allow the cartilage lesion to heal with a type of cartilage that is more like normal hyaline cartilage.

Evaluation

Symptomatic cartilage lesions can present as localized or diffuse knee pain. The knee joint can be viewed as three entities: the medial tibiofemoral, lateral tibiofemoral, and anterior patellofemoral compartments. It is important for the surgeon to localize the patient's symptoms to one or more compartments. With tibiofemoral disease, the pain

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is often worse with weight-bearing activities and located either medial or lateral to the midline, along the tibiofemoral joint line. When the patellofemoral region is involved, patients often complain of anterior knee pain that worsens with activities, such as descending stairs, arising from a chair, or squatting. Recurrent swelling, catching, or locking can also be suggestive of focal chondral defects (Freedman, Fox, & Cole, 2004).

Standard radiographs are the initial imaging modality used for evaluation. They include a weight-bearing posteroanterior image with the knee in full extension, a 45-degree-flexion weight-bearing posteroanterior image, a non-weight-bearing 45-degree-flexion lateral view, and an axial view (also called sunrise or Merchant) of the patellofemoral joint. These views are used to identify joint space narrowing within a single compartment that may be indicative of a focal cartilage lesion or narrowing in multiple compartments, suggesting a more global degenerative arthritic process. Limb alignment and the presence of loose bodies and osteochondral fractures are also assessed. Cartilage is not well visualized on X-ray imaging because it lacks the mineralization of bone, and thus, many focal chondral injuries will have normal plain radiographs. Therefore, if a patient has persistent knee symptoms despite conservative treatment, referral to an orthopaedic surgeon is recommended. Further testing, such as magnetic resonance imaging (MRI), can be useful to better visualize the extent and location of articular cartilage lesions. However, special articular cartilage settings and technique for the MRI are needed, making it more useful for an orthopaedic surgeon to order the study rather than a primary care physician. MRI is not always required, especially because it is not sensitive when looking for focal cartilage injury. When the clinical history and symptoms are consistent with a focal cartilage injury, after plain radiographs are obtained, arthroscopy may be the next indicated step.

Nonoperative treatment of chondral lesions is usually reserved for incidental asymptomatic defects (Freedman et al., 2004). When the lesions become symptomatic, nonoperative treatment is less likely to be successful and operative intervention is warranted (Figure 1).

Surgical Treatment Options

Nonreparative, Nonrestorative

Debridement and Lavage

Debridement and lavage is typically reserved for lower demand older patients with small lesions (<2 to 3 cm^2) and limited symptoms who would have difficulty with activity or weight-bearing restrictions postoperatively (Bert & Maschka, 1989; Federico & Reider, 1997; Freedman et al., 2004; Noyes, Bassett, Grood, & Butler, 1980; Owens, Stickles, Balikian, & Busconi, 2002; Rand, 1991). It entails arthroscopic surgery where two to three small incisions are placed about the knee to place a small camera and instruments inside the joint to evaluate and treat the lesions. Loose chondral flaps that can cause mechanical symptoms are removed. Relief from this type of procedure may be incomplete or temporary because no attempt has been made to restore or repair the cartilage lesion. Current research demonstrates that the best candidates for debridement and lavage are those who suffer from mechanical symptoms (such as a catching or locking sensation when attempting to bend or straighten the knee), which can be caused by a torn meniscus or loose body (Moseley et al., 2002). The recovery time from this type of procedure is relatively short, with immediate full weight-bearing and unrestricted activities.

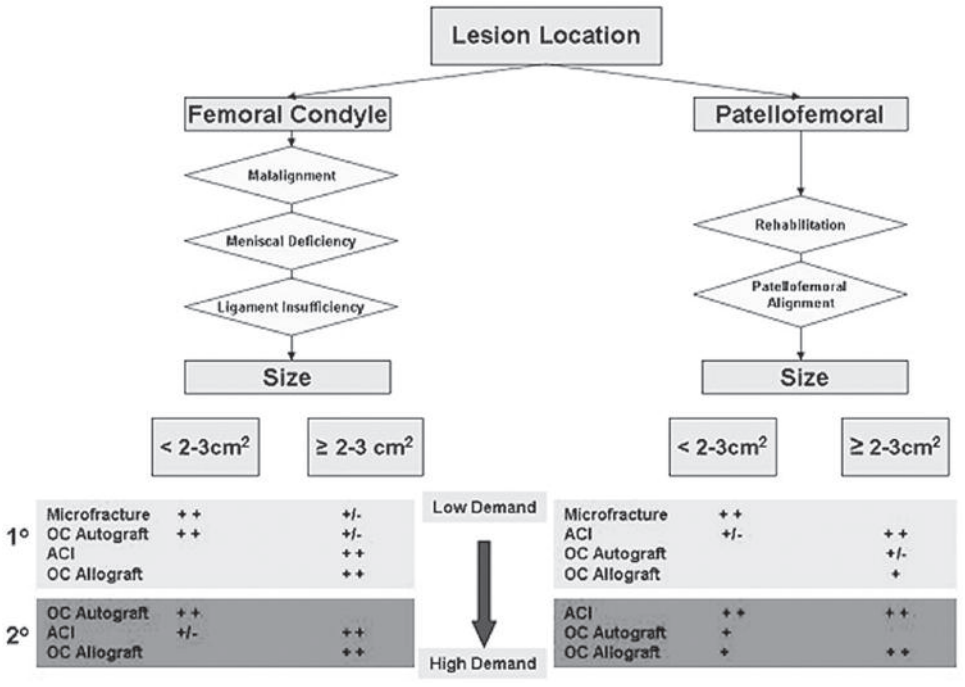

FIGURE 1. The general treatment algorithm which is followed for determining which reparative or restorative technique might be utilized for a specific cartilage lesion.

Reparative

Microfracture

Patients with small to moderate-sized lesions (1 to 5 cm^2) and moderate demands may be treated with marrow-stimulating techniques, such as microfracture. The basis of this treatment option is to stimulate fibrocartilage ingrowth into the chondral defect to cover the underlying bone (Freedman et al., 2004; Gill & MacGillivray, 2001; Steadman et al., 2003). The procedure is done arthroscopically and involves full-thickness cartilage removal down to bone with well-defined sharp boundaries of normal cartilage to prevent injury propagation. An awl is used to create a bed of small holes in the subchondral bone to create bleeding within the bone, allowing cells from the bone marrow to enter the avascular cartilage defect, differentiate into fibrocartilage producing cells, and fill the defect with fibrocartilage (Figures 2A and B). This procedure can be done after failed debridement and lavage or other cartilage-specific surgical procedures.

To achieve optimal results, a strict adherence to postoperative protocol is required. The protocol includes a period of non-weight bearing (4 to 6 weeks) and use of a continuous passive motion (CPM) machine that puts the knee through a specified range of motion without weight bearing. The benefit of CPM is that it prevents arthrofibrosis (or knee stiffness) while the treated cartilage defect is allowed to heal without the stress of weight bearing.

Restorative

Osteochondral Autograft Transplantation

The symptomatic chondral lesion is debrided of cartilage and a small portion of underlying bone to determine the size and shape of the osteochondral autograft that will be needed. Osteochondral autografts are round cylinders of full-thickness cartilage attached to a plug of its underlying bone. These osteochondral autografts are harvested arthroscopically from non-weight-bearing areas of the knee where the articular cartilage and underlying bone can be removed without causing new symptoms or loss of function. The donor graft is inserted using a press-fit technique (Figures 3A and B).

These osteochondral autograft plugs are most commonly transplanted to symptomatic lesions involving the femoral condyles. The lesions should be small to medium-sized (0.5 to 3 cm^2) because the amount of donor tissue available is limited (Freedman et al., 2004; Hangody, Feczko, Bartha, Bodo, & Kish, 2001; Kish & Hangody, 2004). Occasionally, a small incision may be necessary for harvest. For larger lesions, a technique of using multiple plugs called "mosaicplasty" can be employed (Kish, Modis, & Hangody, 1999).

The advantage of osteochondral autografts is that the tissue is autogenous and has normal living hyaline cartilage. Thus, this technique results in cartilage that is most similar to the cartilage that was injured. The disadvantages include donor site morbidity (pain and new cartilage defect), technical difficulty in matching the contour of the defective articular surface to the donor plug, residual gaps between cartilage plugs, and the risk for cartilage or bone collapse. Postoperative recovery requires a period of non-weight bearing and the use of a CPM machine for up to 6 weeks.

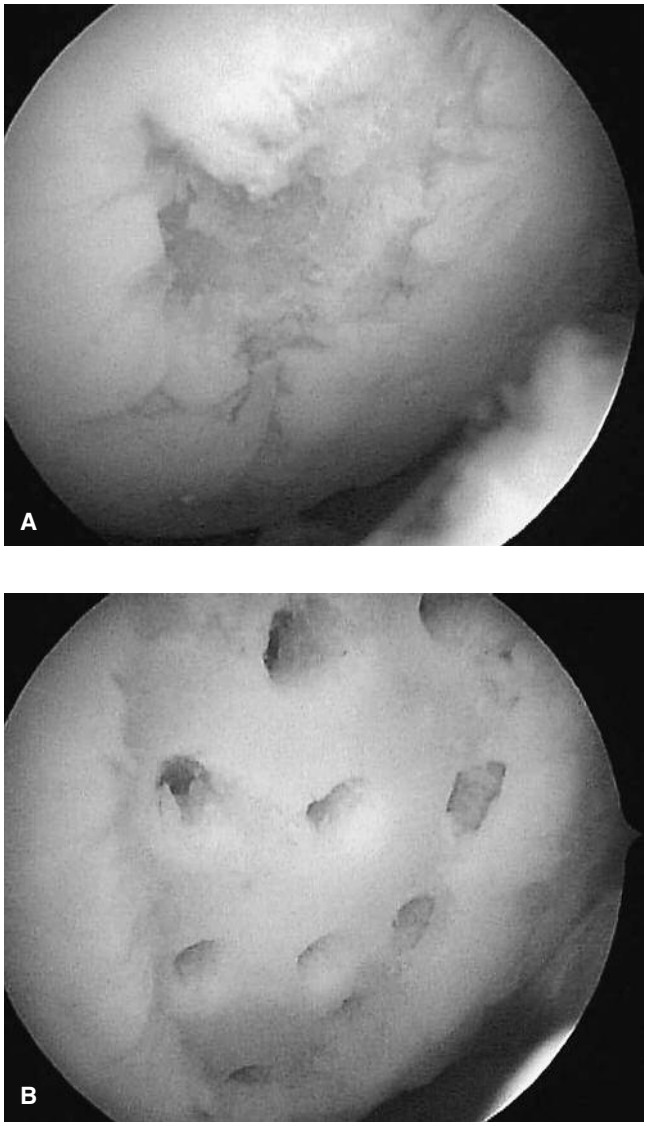

FIGURE 2. (A) The arthroscope is used to visualize a symptomatic focal cartilage lesion involving the femoral condyle before microfracture. **(B)** The femoral condylar lesion has been treated using microfracture technique where vertical boundary walls have been created and small holes have been made to stimulate bleeding and the reparative response.

Osteochondral Allograft Transplantation

Fresh osteochondral allograft transplantation entails the implantation of a cadaveric osteochondral graft into the cartilage defect (Aubin, Cheah, Davis, & Gross, 2001; Bugbee, 2000; Garrett, 1994; Ghazavi, Pritzker, Davis & Gross, 1997; Gross, 1997; Meyers, Akeson & Convery, 1989). A small arthrotomy is made to expose the cartilage defect. An osteochondral allograft plug is harvested to match the defect's size and contour and then press fit to achieve stability (Figures 4A and B).

This procedure is used for medium to large articular cartilage defects (3 cm^2 up to an entire hemicondyle) in older high-demand patients who may have associated bone loss (>6 to 8 mm). Most commonly used for defects involving the femoral condyles, osteochondral allografts may also be used for patella, trochlea, or tibial plateau lesions.

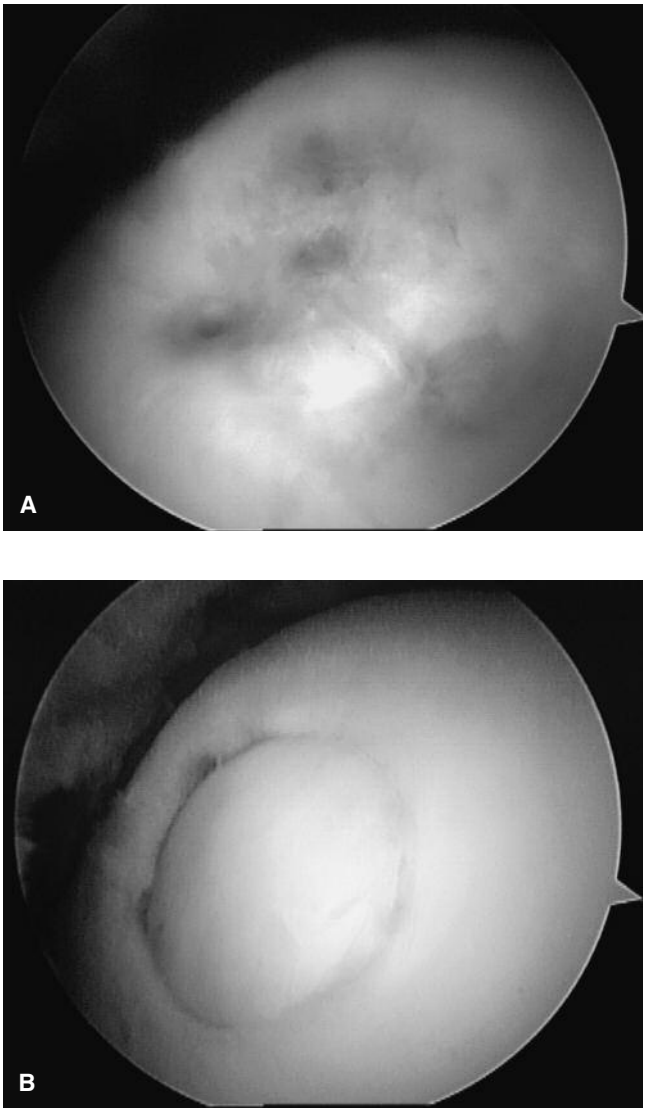

FIGURE 3. (A) The arthroscope is used to visualize a focal cartilage lesion involving the femoral condyle prior to osteochondral autograft transplantation. **(B)** The articular cartilage defect has been debrided, and a portion of the patient's own articular cartilage has been harvested from a non-weight bearing portion of the same knee to fill the defect.

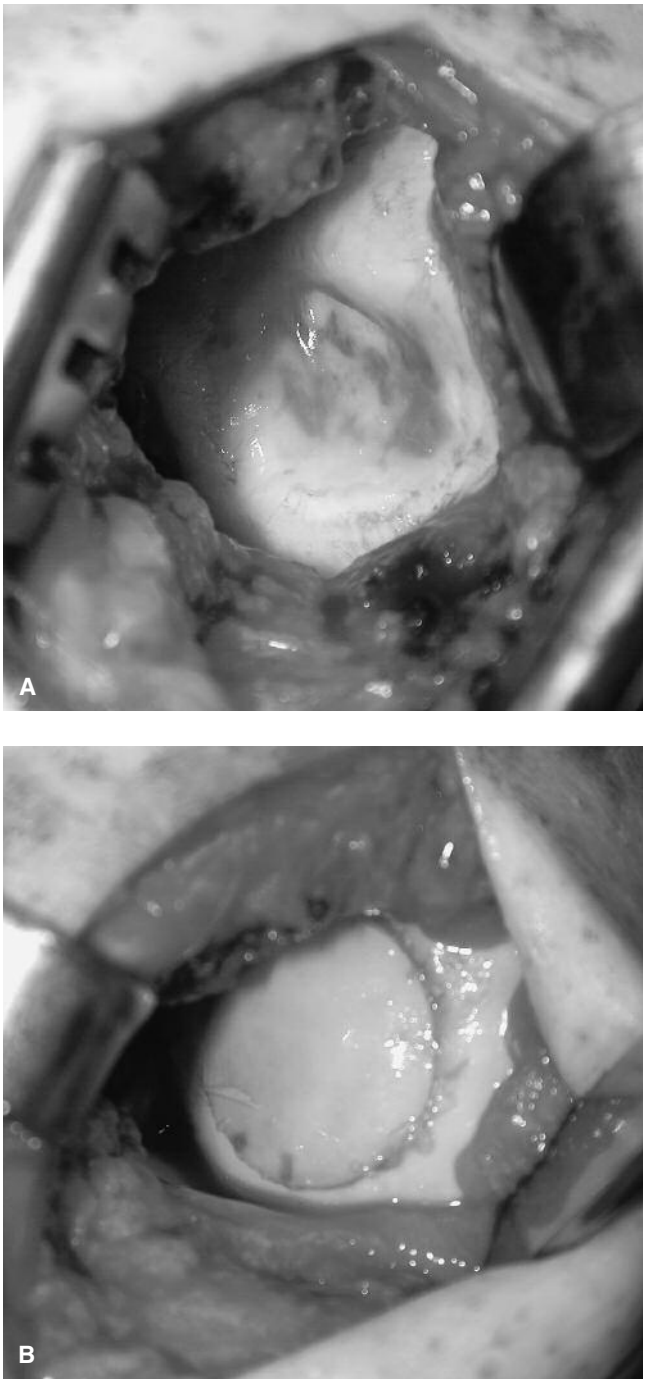

FIGURE 4. (A) A small arthrotomy has been made to expose the articular cartilage defect before osteochondral allograft transplantation. **(B)** The chondral defect has been debrided, and an osteochondral allograft has been inserted to fill the defect.

The major advantage of osteochondral allografts is the ability to replace large osteochondral defects in a single-stage procedure. Additionally, the articular cartilage defect is replaced with articular cartilage rather than fibrocartilage. The disadvantages include graft availability, technical difficulty, cost, and possible disease transmission. Postoperative rehabilitation includes the patient to remain non-weight bearing for 6 to 8 weeks and the use of a CPM machine.

Autologous Chondrocyte Implantation

The procedure is a two-stage technique where during the first surgery, a small piece of cartilage (about the size of a raisin) is harvested arthroscopically from a non-weight-bearing portion of the patient's knee. This cartilage is then sent to a company that processes the cartilage cells. The chondrocytes are isolated and grown in tissue culture to allow them to multiply for several weeks. This results in millions of autologous cartilage cells that are suspended in solution and shipped back to the surgeon for reimplantation. The second stage of the procedure involves a second surgery, an open arthrotomy to expose the lesion, which is débrided, so that the defect has circumferential vertical walls of normal articular cartilage. A periosteal patch is harvested from the ipsilateral tibial shaft to provide a cov-

erage cap over the defect. The patch is sewn in place and sealed with fibrin glue. The chondrocyte-containing solution is then injected into the sealed pouch (Figures 5A and B). The repair tissue that results from this procedure is durable, mechanically firm, and hyaline-like in histology (Peterson, Brittberg, Kiviranta, Akerlund & Lindahl, 2002).

Autologous chondrocyte implantation (ACI) is used for intermediate to high-demand patients who have failed arthroscopic debridement or microfracture. The technique is used for larger (2 to 10 cm^2) symptomatic lesions involving both the femoral condyles and trochlea and the patella (Brittberg et al., 1994; Chu, Convery, Akeson, Meyers & Amiel, 1999; Gillogly, Voight & Blackburn, 1998; Micheli et al., 2001; Minas, 2001; Peterson et al., 2002; Peterson et al., 2000). An intact bone bed is required, making cartilage lesions associated with bone loss better treated by osteochondral grafting.

The postoperative course for this procedure involves non-weight bearing in addition to range-of-motion exercises with the use of a CPM machine for 6 weeks. However, because this involves two surgeries, one of which is a larger open arthrotomy, pain relief and restoration of function may take as long as 12 to 18 months.

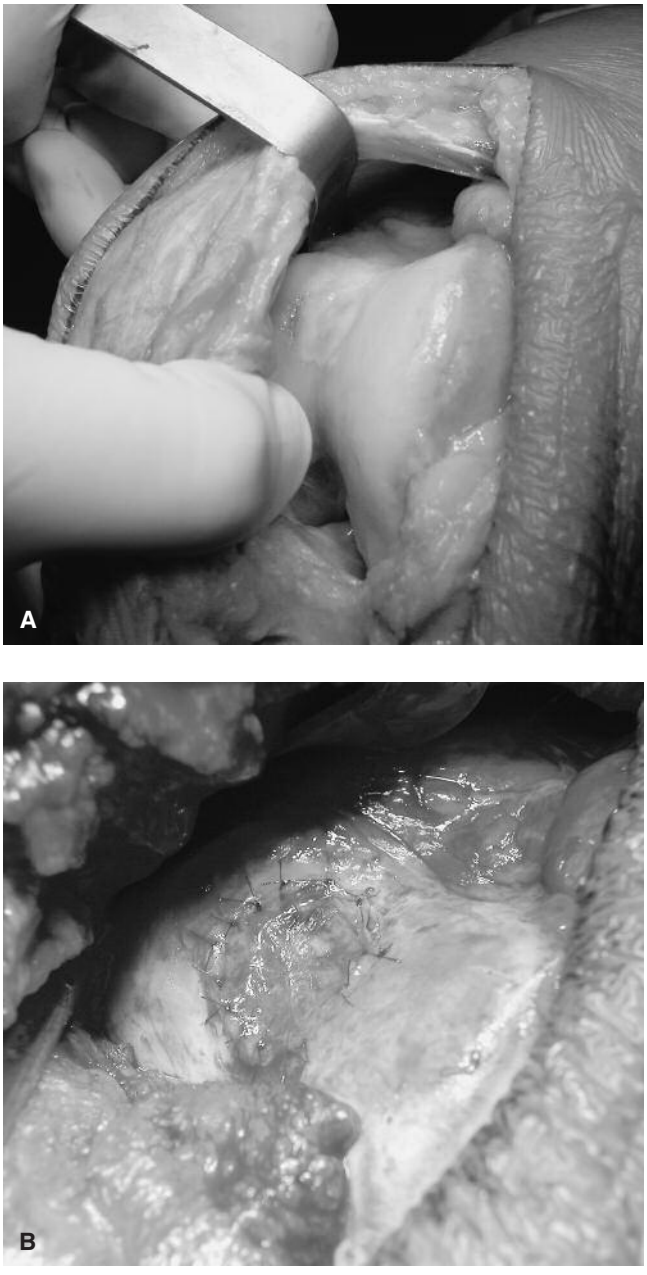

FIGURE 5. **(A)** An arthrotomy has been performed to expose the focal chondral defect on the trochlea prior to autologous chondrocyte implantation. **(B)** The chondral defect on the trochlea has been debrided, a periosteal patch has been harvested from the tibial shaft and sewn in place, and the chondrocytes have been injected inside the created pouch.

Concomitant Procedures

Along with focal chondral lesions, there may be associated injuries or presence of limb malalignment that may also need to be addressed surgically. The most common injuries are ligament and meniscus tears. The meniscus functions as a shock absorber during loading but also distributes force during axial loading. Chondral injuries may occur as a result of a torn meniscus, or a highly irregular chondral surface may predispose a patient to a meniscal tear (Freedman et al., 2004; Messner & Maletius, 1996; Rangger, Klestil, Gloetzer, Kemmler, & Benedetto, 1995; Schimmer, Brulhart, Duff, & Glinz, 1998). If a chondral defect is present in a meniscal-deficient knee, it is paramount that the meniscal deficiency be addressed with a procedure such as a meniscal allograft transplantation (Freedman et al., 2004). In addition, any ligamentous instability must be addressed to restore stability to protect the articular cartilage from excessive shear forces.

If varus malalignment exists with a medial condyle defect, a valgus-producing high-tibial osteotomy should be performed to unload the medial compartment from excessive forces during weight bearing. Similarly, valgus malalignment should be treated with a varus-producing distal femoral osteotomy. In the presence of patellar or trochlear lesions, distal realignment with anteromedialization of the tibial tubercle may be performed primarily to unload the patellofemoral compartment and protect the cartilage repair site (Freedman et al., 2004).

Conclusion

There have been many recent advancements in the treatment of articular cartilage defects of the knee. Reparative procedures, such as microfracture, can provide significant relief of symptoms but do not attempt to recreate the normal articular cartilage. Restorative procedures, such as osteochondral autographs or allografts and autologous chondrocyte implantation, have been developed to recreate the normal articular cartilage surface. The most appropriate option depends on many variables, including both cartilage-specific and patient-specific factors. Thus, a thorough discussion between physician and patient is required to elicit which treatment option is most advisable for each individual.

REFERENCES

Aubin, P. P., Cheah, H. K., Davis, A. M., & Gross, A. E. (2001). Long-term followup of fresh femoral osteochondral allografts for posttraumatic knee defects. *Clinical Orthopaedics, 391*(suppl), S318–S327.

Bert, J. M., & Maschka, K. (1989). The arthroscopic treatment of unicompartmental gonarthrosis: A five-year follow-up study of abrasion arthroplasty plus arthroscopic debridement and arthroscopic debridement alone. *Arthroscopy, 5,* 25–32.

Brittberg, M., Lindahl, A., Nilsson, A., Ohlsson, C., Isaksson, O., & Peterson, L. (1994). Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *New England Journal of Medicine, 331,* 889–895.

Bugbee, W. D. (2000). Fresh osteochondral allografting. *Operative Techniques in Sports Medicine, 8,* 158–162.

Chu, C. R., Convery, F. R., Akeson, W. H., Meyers, M., & Amiel, D. (1999). Articular cartilage transplantation. Clinical results in the knee. *Clinical Orthopaedics and Related Research, 360,* 159–168.

Federico, D. J., & Reider, B. (1997). Results of isolated patellar debridement for patellofemoral pain in patients with normal patellar alignment. *American Journal of Sports Medicine, 25,* 663–669.

Freedman, K. B., Fox, J. A., & Cole, B. J. (2004). Knee cartilage: Diagnosis and decision making. In M. D. Miller, & B. J. Cole (Eds.), *Textbook of arthroscopy* (pp. 555–567). Philadelphia: Saunders.

Garrett, J. C. (1994). Fresh osteochondral allografts for treatment of articular defects in osteochondritis dissecans of the lateral femoral condyle in adults. *Clinical Orthopaedics and Related Research, 303,* 33–37.

Ghazavi, M. T., Pritzker, K. P., Davis, A. M., & Gross, A. E. (1997). Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. *Journal of Bone and Joint Surgery Britain,79,* 1008–1013.

Gill, T. J., & MacGillivray, J. D. (2001). The technique of microfracture for the treatment of articular cartilage defects in the knee. *Operative Techniques in Orthopaedics, 11,* 105–107.

Gillogly, S. D., Voight, M., & Blackburn, T. (1998). Treatment of articular cartilage defects of the knee with autologous chondrocyte implantation. *Journal of Orthopaedic Sports and Physical Therapy, 28,* 241–251.

Gross, A. E. (1997). Fresh osteochondral allografts for post-traumatic knee defects: Surgical technique. *Operative Techniques in Orthopaedics, 7,* 334–339.

Hangody, L., Feczko, P., Bartha, L., Bodo, G., & Kish, G. (2001). Mosaicplasty for the treatment of articular defects of the knee and ankle. *Clinical Orthopaedics and Related Research, 391*(suppl), S328–S336.

Kish, G., & Hangody, L. (2004). A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *Journal of Bone and Joint Surgery British, 86,* 619; author reply 619–620.

Kish, G., Modis, L., & Hangody, L. (1999). Osteochondral mosaicplasty for the treatment of focal chondral and osteochondral lesions of the knee and talus in the athlete. Rationale, indications, techniques, and results. *Clinics in Sports Medicine, 18,* 45–66, vi.

Messner, K., & Maletius, W. (1996). The long-term prognosis for severe damage to weight-bearing cartilage in the knee: A 14-year clinical and radiographic follow-up in 28 young athletes. *Acta Orthopaedics Scandinavia, 67,* 165–168.

Meyers, M. H., Akeson, W., & Convery, F. R. (1989). Resurfacing of the knee with fresh osteochondral allograft. *Journal of Bone and Joint Surgery America, 71,* 704–713.

Micheli, L. J., Browne, J. E., Erggelet, C., Fu, F., Mandelbaum, B., Moseley, J. B., et al. (2001). Autologous chondrocyte implantation of the knee: multicenter experience and minimum 3-year follow-up. *Clinical Journal of Sports Medicine, 11,* 223–228.

Minas, T. (2001). Autologous chondrocyte implantation for focal chondral defects of the knee. *Clinical Orthopaedics and Related Research,* 391(suppl), S349–S361.

Moseley, J. B., O'Malley, K., Petersen, N. J., Menke, T. J., Brody, B. A., Kuykendall, D. H., et al. (2002). A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *New England Journal of Medicine, 347,* 81–88.

Noyes, F. R., Bassett, R. W., Grood, E. S., & Butler, D. L. (1980). Arthroscopy in acute traumatic hemarthrosis of the knee. Incidence of anterior cruciate tears and other injuries. *Journal of Bone and Joint Surgery America, 62,* 687–695, 757.

Owens, B. D., Stickles, B. J., Balikian, P., & Busconi, B. D. (2002). Prospective analysis of radiofrequency versus mechanical debridement of isolated patellar chondral lesions. *Arthroscopy, 18,* 151–155.

Peterson, L., Brittberg, M., Kiviranta, I., Akerlund, E. L., & Lindahl, A. (2002). Autologous chondrocyte transplantation. Biomechanics and long-term durability. *American Journal of Sports Medicine, 30,* 2–12.

Peterson, L., Minas, T., Brittberg, M., Nilsson, A., Sjogren-Jansson, E., & Lindahl, A. (2000). Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. *Clinical Orthopaedics and Related Research, 374,* 212–234.

Rand, J. A. (1991). Role of arthroscopy in osteoarthritis of the knee. *Arthroscopy, 7,* 358–363.

Rangger, C., Klestil, T., Gloetzer, W., Kemmler, G., & Benedetto, K. P. (1995). Osteoarthritis after arthroscopic partial meniscectomy. *American Journal of Sports Medicine, 23,* 240–244.

Schimmer, R. C., Brulhart, K. B., Duff, C., & Glinz, W. (1998). Arthroscopic partial meniscectomy: a 12-year follow-up and two-step evaluation of the long-term course. *Arthroscopy, 14,* 136–142.

Steadman, J. R., Briggs, K. K., Rodrigo, J. J., Kocher, M. S., Gill, T. J., & Rodkey, W. G. (2003). Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. *Arthroscopy, 19,* 477–484.