Chondral injuries

Robert A. Sellards, MD,* Shane J. Nho, BA,‡ and Brian J. Cole, MD, MBA†

Chondral injures are present in up to 10 to 12% of all individuals. When symptomatic, chondral lesions manifest in knee pain, swelling, and loss of function. Cartilage loss may be partial or complete, and it may affect one or multiple locations. The natural history of untreated lesions most likely results in increased disability and progression of cartilage loss. Lesions are classified according to location, depth, and size. Nonsurgical treatment modalities include oral medications, injections, bracing, or physical therapy. Surgical treatment ranges from arthroscopic debridement to implantation of autologous chondrocytes beneath a periosteal patch covering the lesion. The patient's symptoms, age, activity level, and lesion characteristics must be considered and matched with a suitable procedure. Curr Opin Rheumatol 2002, 14:134–141

*Section of Sports Medicine, Louisiana State University Medical Center, New Orleans, Louisiana, USA; †Rush Cartilage Restoration Center, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois, USA; and ‡Rush Medical College, Rush University, Chicago, Illinois, USA.

Correspondence to Brian J. Cole MD, MBA, Rush Cartilage Restoration Center, Midwest Orthopaedics, 1725 West Harrison Street, Suite 1063, Chicago, IL 60657, USA; e-mail: bcole@ortho4.pro.rpslmc.edu

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Abbreviations

ACI	autologous chondrocyte implantation
bRFE	bipolar radiofrequency energy
MRI	magnetic resonance imaging
RFE	radiofrequency energy

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Symptomatic chondral injuries lead to pain, swelling, and disability. Articular lesions occur through sports injury, trauma, osteoarthritis, or osteochondritis. Left untreated, the lesion will most likely remain symptomatic and cause further discomfort. The natural history of untreated asymptomatic articular cartilage injuries is unknown, although once identified as the cause of a patient's symptoms, they rarely spontaneously improve unless treated appropriately.

In the past, surgical options were few ranging from benign neglect to total knee arthroplasty in the advanced stages of osteoarthritis. Today, the orthopaedic surgeon has multiple options available to treat chondral injuries. Techniques range from simple arthroscopic lavage to open procedures that involve autologous chondrocyte implantation. The purpose of this review is to familiarize the physician with the presentation of chondral injuries, illustrate the pathophysiology, and describe the options available for treatment of these debilitating articular lesions.

Epidemiology

Chondral injuries affect nearly one million Americans annually. High-grade lesions are treated with more than 200,000 surgical procedures each year [1]. In one series of 31,516 arthroscopies, Curl and associates identified articular damage in 63% of the patients. Sixty-percent of those affected had high-grade chondral lesions (grade 3 or grade 4 Outerbridge classification) [2]. The most commonly affected zone of articular cartilage damage is the weight-bearing area of the medial femoral condyle (32% of all lesions). Other commonly affected areas include the weight-bearing area of the lateral femoral condyle and patellofemoral joint [3]. These lesions can occur in isolation or they can exist in multiple locations.

Structural conditions that alter knee biomechanics and knee alignment are associated with articular cartilage injury. These include varus or valgus alignment, patellofemoral malalignment, ligamentous instability, and meniscal deficiency. The presence of these conditions must be recognized and treated concomitantly with the articular lesions [4].

Pathophysiology

Normal cartilage

Cartilage has the ability to withstand loads of up to 5 times body weight. Therefore, the goal of treating chondral injuries is to restore this structural integrity [5]. Knee joint cartilage is 2 to 4 mm thick, and is characterized

by being avascular, alymphatic, and aneural. Cartilage is able to provide a low-friction surface and survive repetitive loading in compression, shear, and tension for many decades. It contains a single cell type, the chondrocyte, which secretes an extracellular matrix consisting of proteoglycans. The matrix provides the major resistance to compression and its breakdown is significant in osteoarthritis.

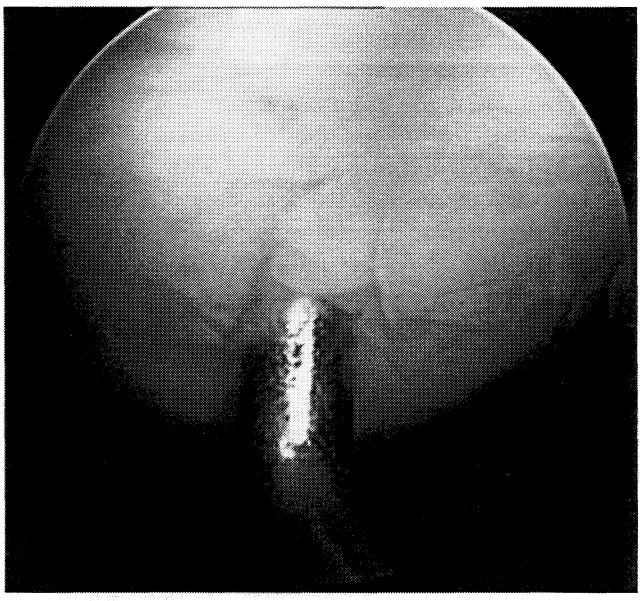
Partial thickness injuries

Partial thickness or superficial defects of articular cartilage fail to heal spontaneously [6–9]. The cell surfaces exposed in partial thickness injuries cannot support cell adhesion, cell migration, or fibrin clot attachment [10]. Matrix proteoglycans containing the glycosaminoglycan dermatan sulfate inhibit cell attachment and clot formation at the site of injury [11,12] (Fig. 1).

Full thickness injuries

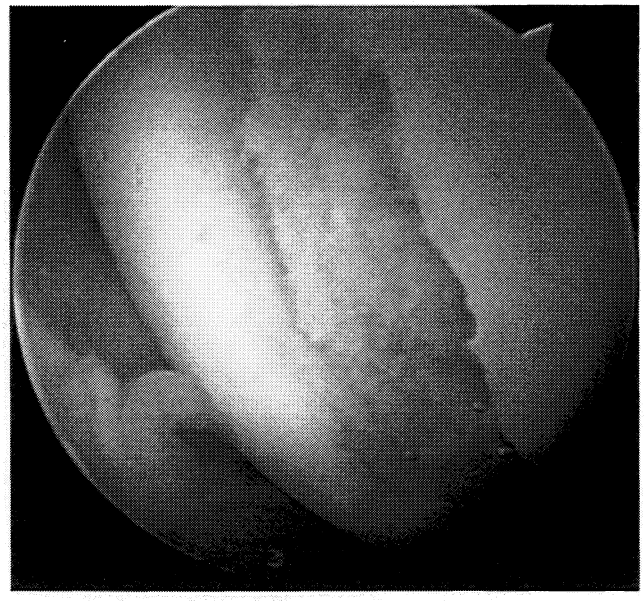
Chondrocytes have limited reparative abilities [13]. If the chondral defect is full thickness, there is a limited spontaneous repair reaction with cells originating from the bone marrow and vascular spaces (Fig. 2) [8,14,15]. In the first few days after injury, fibroblasts and collagen fibrils appear in the clot at the fracture site [11]. In the following two weeks, metaplasia of the mesenchymal cells to chondrocytes progresses and the cells follow with extracellular matrix secretion. The process is completed at 6 months (Fig. 3) [9]. This tissue is not hyaline cartilage, and is characterized by preponderance of Type I collagen rather than the normally abundant Type II collagen potentially accounting for its tendency to degenerate over time [16,17].

Figure 1. Partial thickness articular cartilage defect



An arthroscopic example of a partial thickness articular cartilage defect.

Figure 2. Full thickness articular cartilage defect

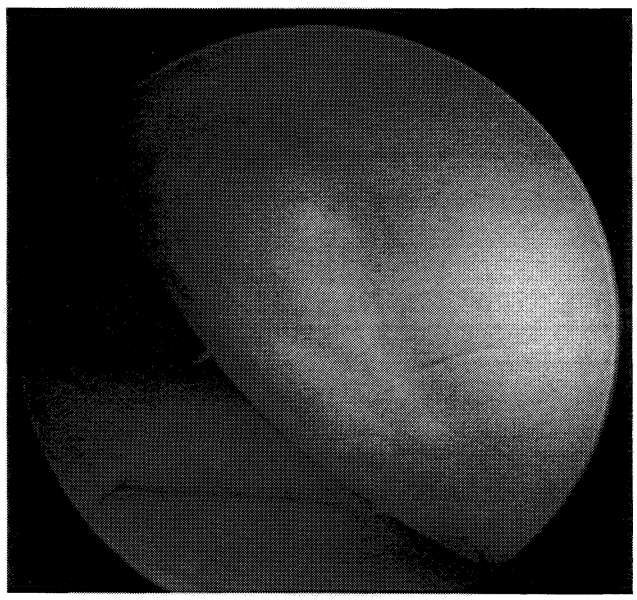


An arthroscopic example of a full thickness articular cartilage defect.

Osteochondral fractures

Osteochondral injuries are more common in adolescents because of weakness of the calcified zone of cartilage next to the subchondral bone (Fig. 4) [18]. Osteochondritis dissecans, a developmental problem involving the subchondral surface, is most commonly seen in the medial aspect of the medial femoral condyle. Alternatively, traumatically induced osteochondral fractures occur most frequently in the lateral femoral condyle and patella [19]. As these defects often involve loss of bone and articular

Figure 3. Fibrocartilage repair tissue



An arthroscopic example of fibrocartilage repair tissue.

is no clinical data showing that these oral agents affect the formation of cartilage [26].

Viscosupplementation is another method of treating chondral injuries, although most research focuses on the overtly osteoarthritic knee. This involves the injection of high molecular weight hyaluronans into the osteoarthritic joint. The two agents available in the United States include Hyalgan (FIDIA S.P.A., Padua [PD], Italy) (sodium hyaluronate) and Synvisc (Biomatrix, Ridgefield, NJ) (hylan G-F 20). Despite the lack of well-controlled studies demonstrating efficacy, viscosupplementation remains an option for conservative treatment of chondral injuries [26] (Table 2).

Table 2. Nonsurgical treatment of chondral defects

- Oral medications
  - Nonsteroidal anti-inflammatory drugs (NSAIDS)
  - Acetaminophen
  - Glucosamine-sulfate
  - Chondroitin-sulfate
- Physical modalities
  - Activity modification: avoidance of high-impact exercises
  - Physical therapy: quadriceps strengthening, hamstring flexibility
- Bracing
  - Knee sleeve for improved proprioception
  - Unloader brace to protect damaged knee compartment
- Injections
  - Corticosteroids
  - High-molecular weight hyaluronans

Surgical modalities

Various operative methods exist for the treatment of cartilage defects. The decision to use these modalities depends on the activity level and age of the patient, as well as the depth, size, and location of the lesion (Table 3).

Table 3. Surgical treatment options for chondral defects

Procedure	Indications	Outcome
Arthroscopic lavage and debridement	Minimal symptoms, short-term relief	Palliative
Radiofrequency energy	Partial thickness defects, investigational	Palliative
Marrow stimulating procedures	Smaller lesions, persistent pain	Reparative
Osteochondral autograft	Smaller lesions, persistent pain	Restorative
Osteochondral allograft	Larger lesions with bone loss	Restorative
Autologous chondrocyte implantation	Small and large lesions with and without bone loss	Restorative
Internal fixation	Osteochondral fragment with bone	Restorative
Genetic engineering	Investigational	Restorative

Internal fixation

In acute osteochondral injuries, the management principle is to use internal fixation to fix the fragment. The goal is to achieve bone-to-bone healing by adequate fixation of the dissociated osteochondral piece. The histologic features of the healed tissue as well as its biologic properties were recently described [19]. At 6.3 years after the index procedure, the tissue examination revealed scarce mature chondrocytes in a regenerative stroma. However, there was congruency of the joint surface in all cases. Fixation should be attempted if the fragment has some remaining bone, is partially attached, and is larger than 1 to 2 cm^2. Results are generally excellent, providing the osteoarticular fragment remains relatively congruous with the surrounding surface with stable fixation.

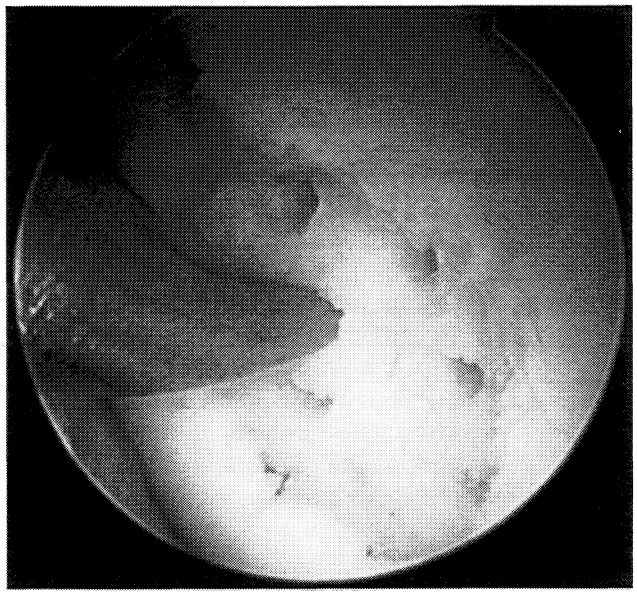
Abrasion arthroplasty and microfracture

Abrasion arthroplasty is performed arthroscopically with a shaver or burr and removes 1 to 2 mm of the exposed sclerotic bone down to the vasculature of the subchondral plate [27]. This allows a clot to form in the defect that may later develop into fibrocartilage. Improvement of joint function and a decrease of knee symptoms have been reported. [28] In a study of failed cartilage repair, the tissue formed after abrasion arthroplasty contained both reparative and degenerative processes; only 2% of the tissue had the appearance of normal articular cartilage [29].

Microfracture involves penetrating subchondral bone to expose the defect to pluripotential marrow stem cells (Fig. 5). These primitive mesenchymal stem cells have the potential to differentiate into chondrocytes. This trait is common to all types of mesenchymal tissue (30,31). Chondrogenic cells include those from the bone marrow, periosteum, cultured periosteal, or perichondrial cells, or from some striated muscle cells [32].

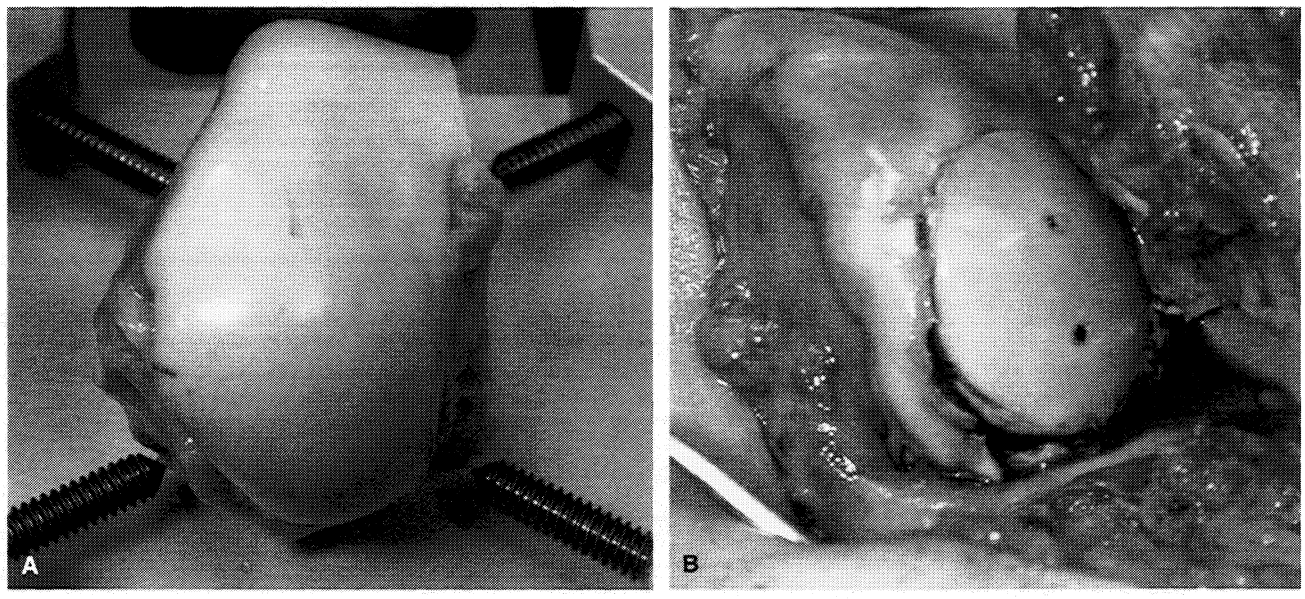
These techniques are recommended for lesions <2 cm^2 in active patients with no more than moderate symp-

Figure 5. Medial femoral condyle lesion



An arthroscopic example of the microfracture technique of a medial femoral condyle lesion.

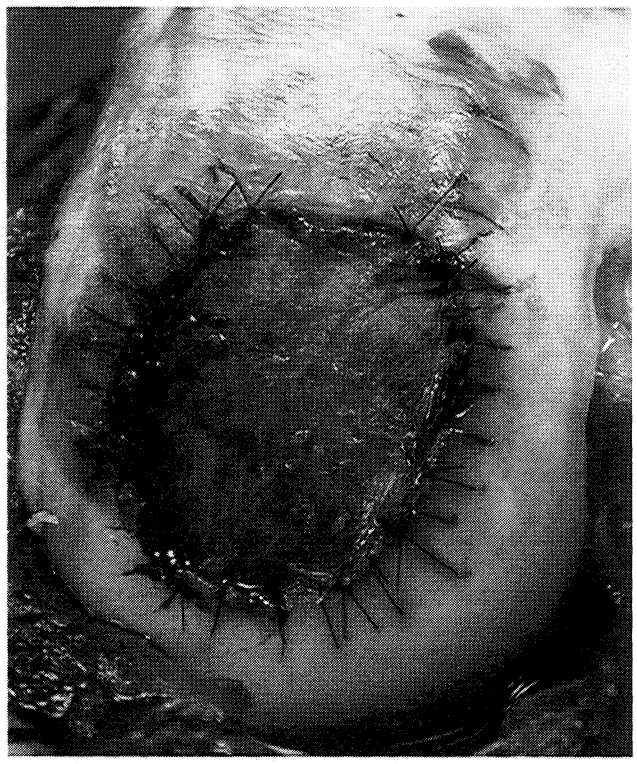
Figure 7. Treatment for a sympotomatic lesion of osteochondritis dissecans



(A) An example of a fresh hemicondyle used to harvest a **(B)** large osteochondral shell allograft to treat a symptomatic lesion of osteochondritis dissecans.

Concerning autologous chondrocytes implantation, Peterson's results on the first 100 consecutive patients with 2 to 9 year follow-up were reported in 1997. There was a high percentage of good to excellent results in patients with single femoral condyle lesions (24 patients, 94%) and in patients with osteochondritis dissecans (19 patients, 89%). The results declined a bit in the presence of a chondral defect with an anterior cruciate ligament injury (16 patients, 75%). There was only a 62% improvement in patients with a patellar graft, but the authors maximized these results when a distal realignment procedure was concomitantly performed. The authors concluded that the ideal candidates for ACI include those with an isolated defect without degenerative changes [51]. This includes active patients with a chondral defect greater than $2cm^2$. Other series of patients report similar results [52].

Figure 8. Defect treated with autologous chondrocyte implantation



An example of a defect treated with autologous chondrocyte implantation.

Genetic engineering

Genetic engineering is a new strategy for treating chondral injuries. This involves a combination of gene transfer techniques and tissue engineering [53]. In gene therapy, specific genes for growth factors are transferred into the chondrocyte or progenitor cells. Once treated, these cells have the potential to produce the growth factors that are conducive to chondrocyte proliferation.

Tissue engineering is based on the creation of biologic substitutes for the repair or regeneration of damaged tissue. The application of this process for chondral defects involves the transplantation of viable cells into an appropriate supportive vehicle. Autologous chondrocyte implantation is an example of this technique, although the ideal scaffold for cartilage engineering has not yet been identified [53].

Summary

The treatment of chondral injuries has significantly advanced over the past two decades. Rather than only providing symptomatic relief with activity modification, or-

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