The Journal of
Maharashtra Orthopaedic Association
March - 2008

Chondral Injuries and Repair

Dr. Vishal Patil,
Dr. Dinshaw Pardiwala.

Arthroscopy & Sports Medicine Service, Department of Orthopaedics,
King Edward VII Memorial Hospital, Mumbai.

The treatment of full-thickness chondral defects in the knee remains a challenge since the goals include not only reestablishing normal anatomical articular surface contours, but also normal biomechanical and histologic integrity. Untreated articular cartilage lesions have little or no potential to spontaneously heal with normal hyaline cartilage. Although these lesions may be small and asymptomatic at the time of discovery, they may increase in size and become painful at a later date if left untreated. Besides, evidence suggests that chondral damage in the knee predicts early development of osteoarthritis.

❖ Anatomy ❖

The structural components of hyaline cartilage are chondrocytes, collagen, extracellular matrix proteoglycans, noncollagenous proteins, and water. Chondrocytes constitute the basic building block of the articular surface and are the major source for new synthesis and maintenance of its components. Undifferentiated mesenchymal marrow stem cells propagate through the calcified cartilage zone to become chondroblasts which become isolated in lacunae, and form chondrocytes. These cells receive their nutritional support from the synovial fluid. In skeletally mature articular cartilage, chondrocytes no longer divide but still remain alive, whereas in skeletally immature articular cartilage chondrocytes undergo cell division and increase cell matrix volume. As chondrocytes age, they exhibit a decrease in cellular activity, especially production of both collagen and proteoglycan.

Water is one of the most important components of cartilage and constitutes approximately 75% of the weight of articular cartilage. Collagen, redominantly type II, underlies the form and tensile strength of articular cartilage. Proteoglycans, with their structural subunits, glycosaminoglycans, trap and hold water within cartilage thus provide the compressive strength of articular cartilage.

The microarchitecture of articular cartilage is unique. The outermost layer, or superficial zone, lamina splendens, which contains a relatively small amount of proteoglycan, is thin, noncellular, and porous. In this layer the fibers are arranged parallel to the joint surface. In the middle zone, the collagen fibrils have a larger diameter compared with those in the superficial zone, with a higher concentration of proteoglycans and lower amounts of water and collagen. In the third layer, or deep zone, the largest-diameter collagen fibrils, the highest concentration of proteoglycans, and the lowest concentration of water are noted. This microanatomy reduces the forces of friction across the joint to extremely low values. Recreating this complex microstructure makes surgical reconstruction of articular cartilage very difficult.

Articular cartilage lacks vascular, neural, and lymphatic access networks, which creates a limited environment for spontaneous repair. Injuries that do not penetrate into the subchondral bone show little sign of spontaneous repair, whereas those that extend into the depth of subchondral bone initiate a vascular proliferative response that produces only fibrocartilage.

❖ Clinical Features ❖

The most common clinical presentation of a full-thickness articular cartilage lesion is a loose body.

may have an insidious onset with no effusion, or may be associated with an acute injury, with a concomitant large knee effusion. Other patients may have a progressive onset of joint-line and/or patellofemoral pain with occasional mechanical symptoms of locking or catching. Common scenarios for the presentation of full-thickness articular surface injuries include patellar dislocation with lateral femoral condyle and medial-patella facet lesions.

The physical examination usually does not elicit any consistent finding other than localized pain with or without an effusion. The presence of a loose body should be considered predictive of the occurrence of an articular surface injury until proven otherwise.

❖ Imaging ❖

Plain radiographs may visualize compartment joint-space narrowing or an osteochondritis dissecans, with or without a loose body [Fig. 1]. With full-thickness articular cartilage lesions, plain radiography might not reveal any changes; in that setting, magnetic resonance (MR) imaging may be more helpful. Proton-density imaging of thin sections and T2-weighted imaging with fat-saturation sequences optimize resolution of the articular chondral surface [Fig. 2]. High-resolution gradient-echo imaging has also been proposed to allow more careful evaluation of the articular surface of the patella. Defects are best analysed in 3 planes.

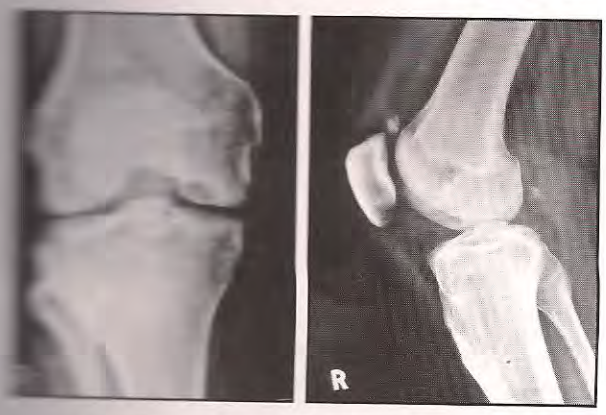

Fig. 1 : Classical osteochondritis dissecans of medial femoral condyle with a loose body.

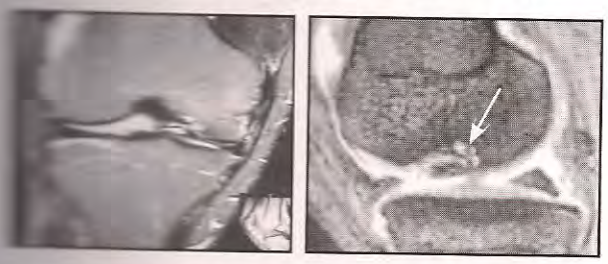

Fig. 2 : MRI optimizes resolution of the articular chondral surface.

The role of the bone scan remains controversial. Isolated articular surface defects that do not penetrate the subchondral bone might not be identified by bone scanning.

❖ Documentation of Arthroscopic Findings ❖

The Outerbridge system for grading the status of articular cartilage, is the simplest working tool for describing the condition of the joint and the nature of a chondral lesion. In grade I, the articular surface is swollen and soft and may be blistered. Grade II is characterized by the presence of fissures and clefts measuring less than 1 cm in diameter. Grade III is characterized by the presence of deep fissures extending to the subchondral bone, measuring more than 1 cm in diameter. Loose flaps and joint debris may also be noted. In grade IV, subchondral bone is exposed [Fig. 3].

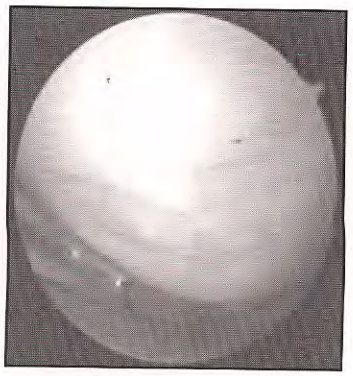

Fig. 3 : Grade IV Outerbridge chondral defect.

This must be combined with an accurate notation of the location, size (surface area), and shape (circular, rectangular, or elliptical) of the articular surface lesion and a description of the walls (contained, partially contained, or open). The depth of the lesion designated as partial thickness, full thickness, or extending into subchondral bone may be the major determinant in the final selection of the surgical technique to be utilized.

❖ Nonoperative Treatment ❖

The goal of nonoperative treatment is to reduce symptoms related to the articular cartilage lesion, not to restore anatomy. Physical therapy for muscle strengthening, gait training, and application of appropriate bracing or use of an orthotic device may eliminate some of the symptoms. Use of intra-articular viscosupplementation products and oral chondroprotective agents for the treatment of osteoarthritis may also provide symptomatic relief, but to date there has been no evidence of structural improvement.

❖ Surgical Options ❖

The various techniques available for surgical intervention result in a tissue response that is either reparative or restorative. The chondrocytes for all of these procedures are facilitated from mesenchymal stem cells induced from periosteum or perichondrium, harvested as autologous chondrocytes, or transplanted as allogeneic chondrocytes. The goal of restorative surgical techniques is complete reconstruction of the microarchitecture of articular cartilage, with restoration of all biomechanical and physiologic functions and resultant complete relief of symptoms. In contrast, a reparative surgical technique reconstructs the defect in a manner that does not necessarily restore the articular cartilage architecture but still may relieve symptoms. There are also some operative techniques that have no impact on the articular cartilage defect itself. For example, arthroscopic lavage and debridement (chondroplasty) may lessen symptoms, but the effects diminish with time. Patients with angular deformity and articular surface lesions (generally due to osteoarthritis) may show signs of clinical improvement and increased joint-space widening after osteotomy; however, biopsy specimens obtained from the arthritic compartment consistently show proliferation of a fibrocartilaginous response with little hyalinelike cartilage restoration. Similarly, varus or valgus bracing may offer symptomatic relief to the patient with a malaligned knee without changing the damaged articular surface structure.

Truly restorative procedures for the treatment of full-thickness articular surface lesions are limited to single-plug osteochondral autograft transfer and osteochondral allograft reconstruction. The other available procedures attempt to achieve full restoration of only the articular surface and therefore should be considered merely reparative.

Arthroscopic debridement : Arthroscopic debridement (chondroplasty) to remove loose flaps or edges that mechanically impinge on the joint will temporarily improve symptoms.

Abrasion arthroplasty : Abrasion arthroplasty is indicated in the treatment of an exposed sclerotic degenerative arthritic joint lesion. It involves careful intracortical superficial abrasion to create a vascular response not mediated by the subchondral bone marrow elements, but rather by cells within the joint itself. Reparative tissue with predominantly fibrocartilaginous responses appears to be the dominant result of this technique.

Microfracture techniques : Microfracture techniques, such as drilling of sclerotic subchondral exposed bone, stimulate the formation of a smooth fibrocartilaginous surface. Alternatively surgical awls [Fig. 4] may be used to create several subchondral puncture holes 3 to 4 mm apart so as to eliminate heat generation. Important technical adjuncts are careful debridement of the calcified cartilage layer and the use of postoperative continuous passive motion (CPM) with protected weight bearing for 6 to 8 weeks.

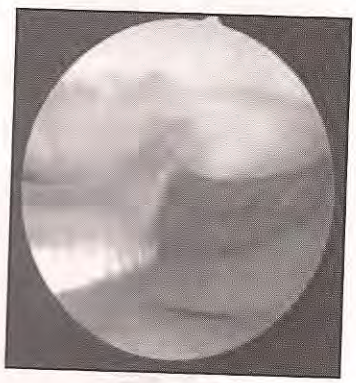

Fig. 4 : In the microfracture technique, surgical awls are used to create several subchondral puncture holes 3 to 4 mm apart so as to stimulate the formation of a smooth fibrocartilaginous surface.

Periosteal and perichondrial grafting : Periosteal and perichondrial grafts have been demonstrated to effect chondroneogenesis in vitro from their cambrium layer.

Autologous chondrocyte implantation (ACI) : Autologous chondrocyte implantation was first reported in 1994 by Brittberg. They initially harvested autologous chondrocytes from patients and then expanded and manipulated these cells in culture, prior to reimplantation under a periosteal flap (Fig. 5). Second-look arthroscopy and biopsy for treated femoral lesions revealed hyaline-like tissue repair in a majority of lesions. ACI is indicated for the younger (aged 20 to 50 years) active patient with an isolated traumatic femoral chondral defect greater than 2 to 4 cm. Care should be taken to ensure that the lesion is not so deep (i.e., 3 to 6 mm into the subchondral boundary) that an initial repair of the subchondral base might be necessary. Accompanying ligamentous and meniscal lesions, joint malalignment, and patellofemoral instability must be corrected concurrently. Absence of a meniscus and bipolar lesions of the articular surface are contraindications to ACI. Newer generations of ACI do not require a periosteal cover, and in these the cultured chondrocytes are supplied impregnated on a resorbable collagen matrix (MACI).

Fig. 5 : Autologous chondrocyte implantation involves an arthroscopic articular cartilage biopsy from patients, followed by in vitro cell culture and expansion, and its reimplantation under a periosteal flap.

Osteochondral autograft transfer : Osteochondral autograft was first reported by Judet for treatment of osteochondritis dissecans defects in the femur. The lateral patellar facet was used as an autograft and upto one third of the articular width was removed. A follow-up study of 10 patients an average of 6.5 years after the procedure revealed satisfactory functional results with decreased symptoms, however, postoperatively, most patients had donor-site patello-femoral pain.

The mosaicplasty procedure [Fig. 6] popularized by Hangody provides treatment options for much larger and deeper femoral condylar or patellar defects. The technique is dependent on surgical skill to recreate the normal radius of curvature in the femoral condyle, and involves using multiple small osteochondral peg grafts (2.7 to 10 mm in diameter) to press-fit together a repair of a large defect. The cylindrical autografts are harvested arthroscopically from the lateral aspect of the lateral femoral trochlea superior to sulcus terminalis, Second-look arthroscopic biopsies have confirmed

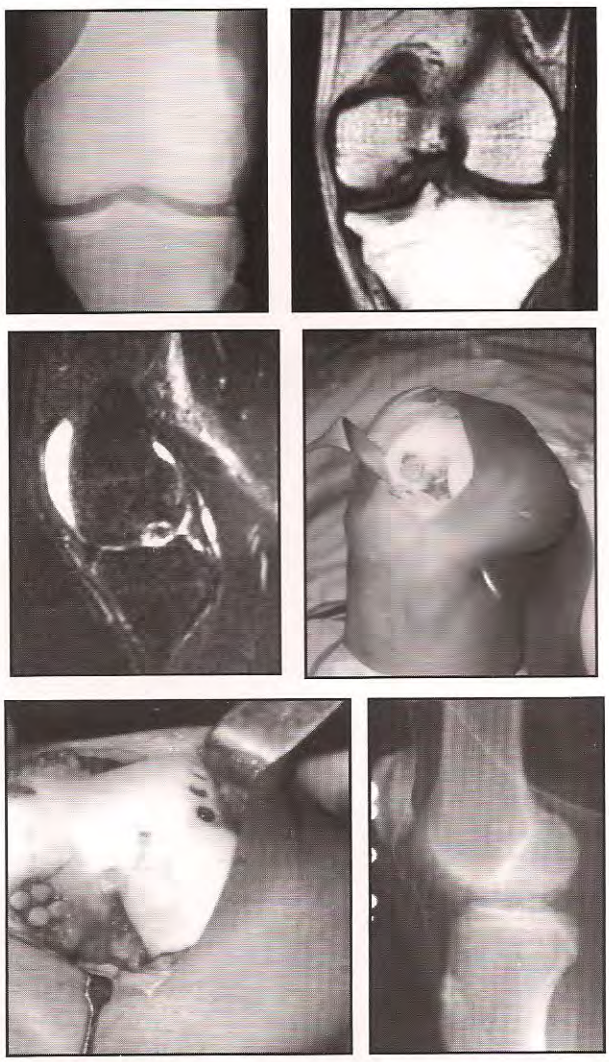

Fig. 6 : Mosaicplasty for a large osteochondritis dissecans of the medial femoral condyle.

that the transplanted cartilage remains hyaline in character and that donor-graft bonding sites are fibrocartilaginous. The use of autografts is appealing; however, there is a limited amount of donor-graft tissue available for transfer and a potential risk of donor-site morbidity [Fig. 7]. The two-dimensional surface area can be covered with this technique, but it is difficult to reproduce the three-dimensional contour of the femoral condyle. Collapse of the osteochondral dowels by migration or degradation leads to flattening in the area of the mosaicplasty.

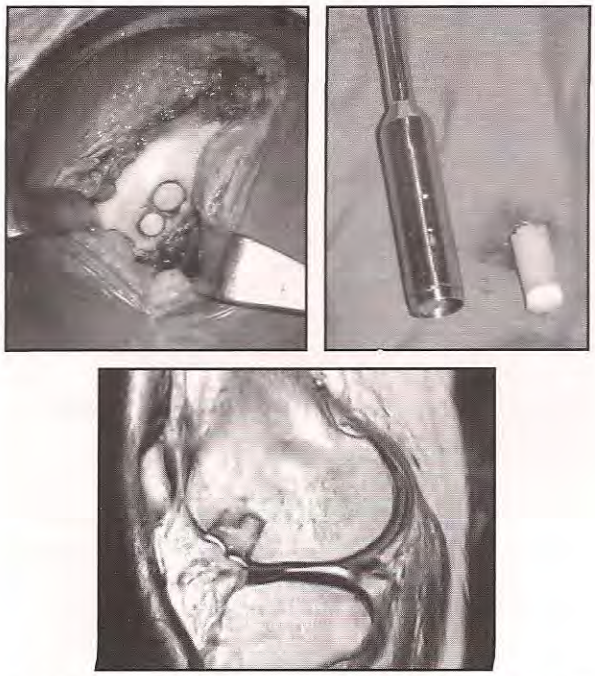

Fig. 7 : Osteochondral autograft transfer is limited by the amount of donor graft tissue available for transfer and large reconstructions have a potential risk of donor-site morbidity including patellofemoral pain.

Osteochondral allograft transplants : Osteochondral allografts may be used for larger (> 4 cm^2) full-thickness lesions after the failure of one or two previous surgical procedures [Fig. 8]. Fresh allografts (i.e., obtained within 24 to 72 hours) provide the greatest likelihood of chondrocyte survivability, but also carry a higher risk of immunogenic and transmissible disease. Use of a shell graft (one with < 1 cm of subchondral bone base) reduces immunogenicity of the graft by decreasing exposure of white cells found in cancellous bone. A factor contributing to the failure of osteochondral allografts is the host-directed tissue remodeling of the graft by creeping substitution. The technical constraints of surgical implantation of fresh osteochondral allografts are extremely demanding. Fresh tissue from a young donor (< 40 years old) must be available; the recipient patient must be on call; and the surgeon must be able to transplant the graft at all hours of the day and night.

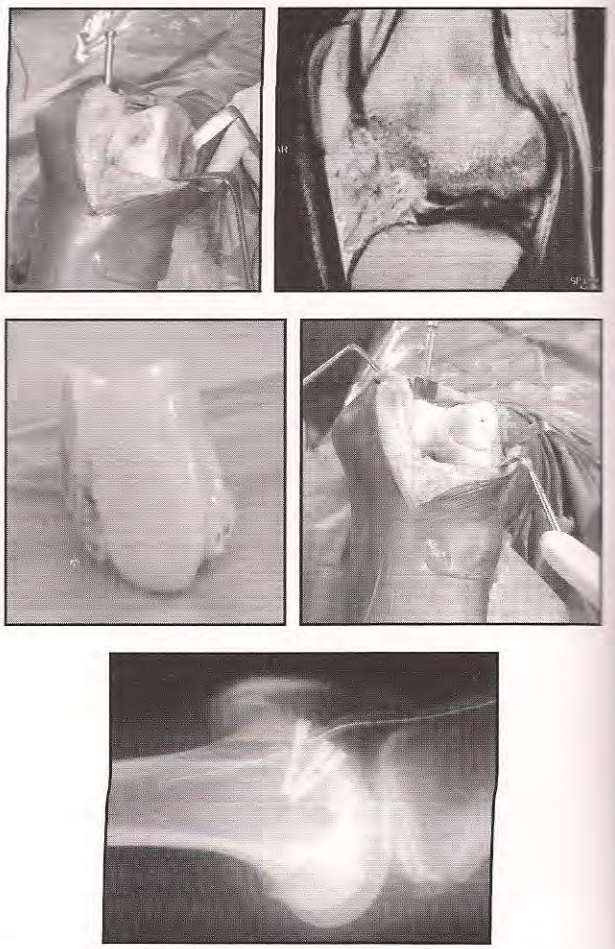

Fig. 8 : Osteochondral allograft transplantation for an extensive osteochondral defect of the femoral trochlea in a young male.

The best indication for the procedure are a post-traumatic or osteochondritis dissecans defect. A total of 126 procedures on 123 knees were reviewed at an average follow-up interval of 7.5 years. The success rate (defined on the basis of achieving good or excellent results) at 5 years was 95%; at 10 years, 71%; and at 20 years, 66%.

Details of osteochondral allograft transplants are described in a subsequent chapter.

❖ Rehabilitation ❖

All of these procedures except debridement require protected weight bearing for varying time periods (a minimum of 6 weeks). Allograft techniques usually require longer periods of protected weight bearing (3 to 4 months). Continuous passive motion may be helpful for improving surface contour during the postoperative period. Return to functional work and sports activities is possible with all the procedures.

A Practical Algorithm of Surgical Options

Prior to treating full-thickness articular surface lesions the following prerequisites should be satisfied:

1. Age range from skeletal maturity to 50 years.
2. Stable knee ligaments, with either preoperative or concurrent reconstruction of any deficiency.
3. Stable neutral tracking extensor mechanism.
4. Intact menisci (meniscal allograft may be necessary).
5. Single or multiple full-thickness femoral condyle or patellar articular surface defects without bipolar defect (i.e., femoral tibial and/or patellofemoral joint-surface changes greater than grade 2.
6. A defect that is not osteoarthrotic or associated with inflammatory joint.

Figure 9 outlines a simplified approach to decision making in the surgical treatment of full thickness chondral defects. Anterior femoral condyle, femoral trochlear, patellar, and tibial anterior third full thickness chondral lesions less than 2 cm in diameter are ideally suited for mosaicplasty. Well shouldered lesions less than 5 mm in diameter should be treated with debridement, and microfracturing. Microfracture revascularization is also an option for very large defects at sites not amenable to mosaicplasty, and for bipolar lesions. Traumatic joint injuries that have resulted in loss of surface contour involving a significant extent of the compartment are best handled with fresh allograft even as a primary surgical procedure.

❐ ❐ ❐

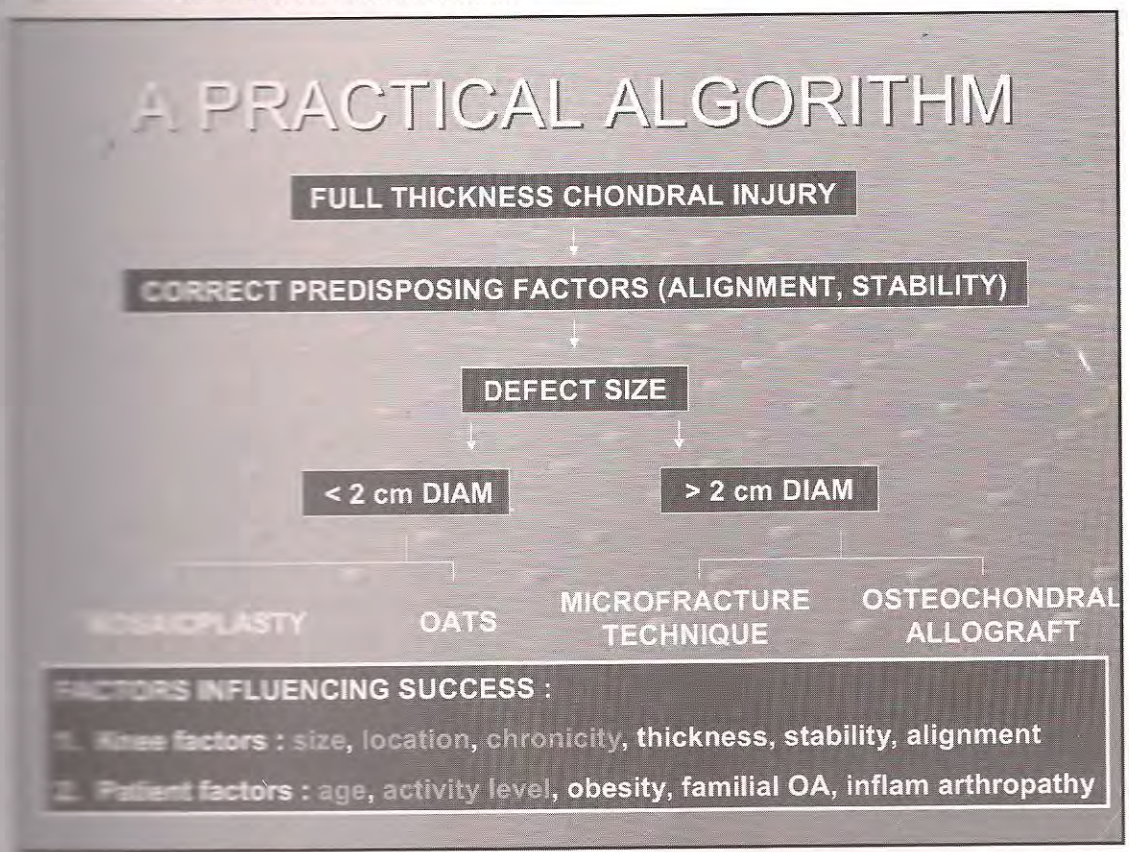

Fig. 9: A practical algorithm to decision making in the surgical treatment of full thickness chondral defects.