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Management of Knee Articular Cartilage Injuries

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1. Introduction

Articular cartilage is a unique, biologically active tissue. In the knee, it serves as the end-bearing surface for the distal femur and proximal tibia, forming a diarthrodial synovial joint capable of enduring years of impact loading. Made of hyaline cartilage, the near-frictionless surface distributes load throughout motion across 6 degrees of freedom, reducing stress transmission to the underlying subchondral bone. This permits weight-bearing during both activities of daily living and high-impact athletics. In fact, forces in the knee joint may approach 8-times body weight during deep knee bends(Reilly and Martens 1972) and pressures up to 12 MPa during maximal quadriceps contraction(Huberti and Hayes 1984) (a pressure equivalent to being ¾ mile under water). These biomechanical properties rely on compression and deformation, a direct result of the biphasic nature of articular cartilage consisting primarily of water and extracellular matrix. Variation in this tissue's thickness and the joint's radii of curvature further influence biomechanics because of compartmental-specific loading profiles within the tibiofemoral and patellofemoral "joints" of the knee.

Arthroscopic appearance of articular cartilage in the knee (Figure 1a) should display a glistening, smooth, white surface that is firm to manual or instrumented palpation. Any disruption in the smooth surface or the tactile feel is abnormal. The loss of articular cartilage is the *sine qua non* of osteoarthritis. Histologic examination reveals relatively hypocellular tissue that lacks a vascular supply, neural input and output, and lymphatic drainage. These features contribute to the minimal innate healing response of isolated chondral damage and also illustrate the difficulty in making a clinical diagnosis of an isolated chondral defect (Figure 1b). Further, the role of the subchondral bone and its complex interaction with the overlying layered structure of cartilage has been emphasized in recent literature, not only in defect creation and progression, but also in surgical treatments. At the current time, these surgical procedures, cartilage repair and restoration, are designed to prevent and/or delay the initiation and/or progression of osteoarthritis.

2. Anatomy and biomechanics

The knee joint is the largest in the human body. It is a modified hinge allowing motion in the flexion / extension (sagittal), varus / valgus (coronal), and internal / external rotation planes (axial). These motions have both osteocartilaginous and soft tissue ligamentous constraints. The patella articulates with the femoral trochlea and the medial and lateral femoral condyles articulate with the medial and lateral menisci and tibial plateaus. The

collateral (medial and lateral collateral, MCL and LCL) and cruciate (anterior and posterior cruciate, ACL and PCL) ligaments are restraints to abnormal motion in one or more planes.

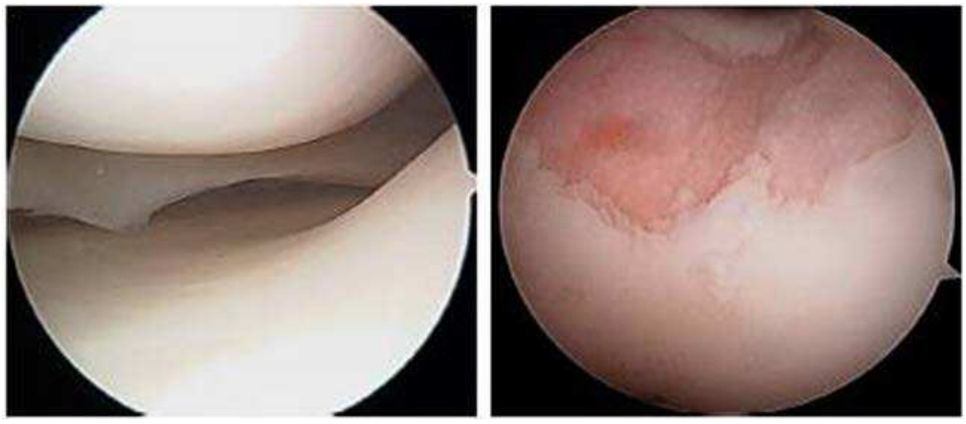

Fig. 1. 1a) Arthroscopic photograph of normal knee articular cartilage demonstrating smooth, white, glistening surface; 1b) Arthroscopic photograph of isolated, full-thickness chondral defect of femoral trochlea.

2.1 Tibiofemoral compartments

Articular cartilage in the tibiofemoral joint articulates with both meniscus and opposing surface articular cartilage. The menisci increase surface contact area, thus reducing stress transmission to the under- or over-lying articular cartilage. Both anatomy and kinematics are significantly different within each of the tibiofemoral compartments(Iwaki, Pinskerova et al. 2000). This asymmetry is reflected in that the lateral compartment tends to axially rotate around a relatively stationary medial compartment with knee flexion.

Kinematically, the medial compartment of the knee operates like a ball (femoral condyle) and socket (tibial plateau and meniscus)(Scott 2005). In the sagittal plane, the medial femoral condyle is composed of two arcs of different radii of curvature and the medial tibial plateau of two angled flat surfaces(Iwaki, Pinskerova et al. 2000). The more anterior surface (extension radius / facet) of the femur has a larger radius than that of the posterior surface (flexion radius / facet). The tibia's angled flats, together with the firmly-attached medial meniscus, create a concavity in which the femoral condyles contact.

Contrary to the medial side, the lateral compartment of the knee has a convex-to-convex articulation in the sagittal plane (Figure 2). Without a lateral meniscus, the lateral compartment operates via nearly point-on-point contact. With a single radius of curvature, the femoral condyle tends to roll back on the tibial plateau, which supports a more loosely-attached lateral meniscus, with knee flexion(Iwaki, Pinskerova et al. 2000). The fixed axis medially combined with greater mobility laterally supports the "screw-home mechanism" of tibial internal rotation with increasing knee flexion(Blankevoort, Huiskes et al. 1988).

Similar to the anatomic asymmetry between the medial and lateral compartments, the biomechanical loading profiles are also unique. In a normal knee, the lateral meniscus covers a greater surface area (~80%) of the plateau than the medial (~60%)(Clark and Ogden 1983), thus transmitting a larger proportion of the axial load while weight-bearing (50% medially versus 70% laterally)(Fukubayashi and Kurosawa 1980; Ahmed and Burke 1983). Following meniscectomy, all load is transmitted through the articular cartilage and the

femur – tibia geometrical asymmetry medially versus laterally plays a greater role. This is reflected by the nearly 300% increase in contact stress laterally versus 100% increase medially after total meniscectomy(Kettelkamp and Jacobs 1972; Fukubayashi and Kurosawa 1980). These findings clearly illustrate the chondroprotective role of the menisci in the knee (Figures 3a, 3b).

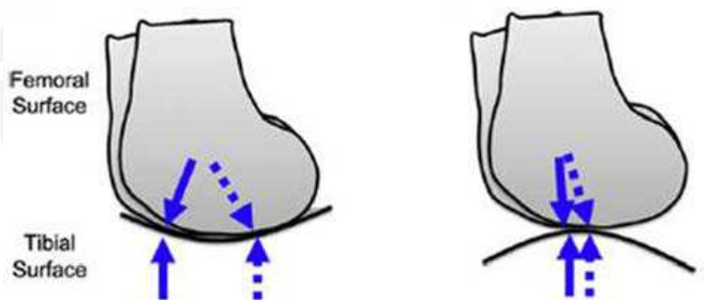

Fig. 2. The "ball-in-socket" schematic of the medial compartment articulation (left); the less congruent, convex-on-convex articulation of the lateral compartment (right) (reproduced with permission from Koo S, Rylander J, Andriacchi T: Knee joint kinematics during walking influences the spatial cartilage thickness distribution in the knee, in *Journal of* Biomechanics 2011; 44(7): 1408. Publisher Elsevier).

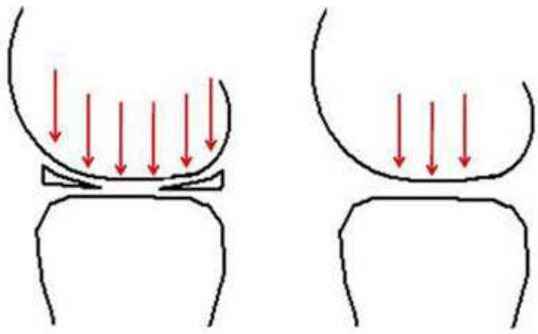

Fig. 3. 3a) Sagittal profile of knee with normal menisci. Axial load distributed across surfaces of menisci and articular cartilage; 3b) Sagittal profile of knee without meniscus. Axial load distributed over articular cartilage only. With same force and smaller area of articulation, increased stress transmitted to articular cartilage.

2.2 Patellofemoral compartments

The patellofemoral articulation consists of the patella, the largest sesamoid in the human body, and the trochlea, a groove located on the anterior distal femur. The patella normally sits within a suprapatellar pouch in full extension and begins to engage the trochlea around 20 to 30 degrees of knee flexion. A vertically-oriented ridge on the articular surface of the patella separates the patella into medial and lateral facets. The superior 75% of the patella is articular cartilage, while the inferior 25% is non-articulating bone. The thickness of the articular cartilage in the patella can be the thickest in the human body, up to 5- or 6-mm (Scott 2005). This portends the ability to withstand high joint reactive forces seen in the patellofemoral articulation. The trochlea is separated into medial and lateral facets by a

vertically-oriented trough that continues inferiorly into the intercondylar notch. The lateral facet of the trochlea extends anteriorly slightly more than that of the medial facet, providing a lateral buttress to patellar instability. The bony articular congruity attained by the patella and trochlea provides inherent static stability to the patellofemoral articulation.

The biomechanics of the patellofemoral joint are dependent upon both bony and soft tissue constraints. The patella engages the trochlea at approximately 20 degrees of knee flexion. At this position, the medial patellofemoral ligament functions as a primary restraint to lateral patellar translation(Conlan, Garth et al. 1993). With increasing flexion, the patella contacts the trochlea via a horizontal area of contact. Near extension, the inferior articular surface of the patella is "articulating." With increasing flexion, the horizontal contact area moves further proximal on the patella until this area is divided into two separate areas of contact on the medial and lateral femoral condyles at around 120 degrees of flexion. Contact pressure in the patellofemoral joint is greatest between 60 and 90 degrees of flexion, with maximum pressures of up to 12 MPa attained during forceful extensor mechanism quadriceps contractions(Huberti and Hayes 1984).

2.3 Microscopic anatomy

The microscopic composition of articular cartilage appears as a highly-organized, layered system of cells and extracellular matrix (ECM). The chondrocyte is the only cell present in articular cartilage and occupies only 5% of its total volume(Lieberman 2009). Thus, this cell is exclusively responsible for maintenance of the ECM. It receives its nutrition from synovial fluid diffusion from the interior of the joint. Embedded within the ECM, the chondrocyte is relatively immunoprivileged. This isolation also accompanies a lack of vascular or nerve connections, or lymphatic drainage. Thus, cartilage has a limited innate healing capacity.

Articular cartilage can be broadly grouped into two separate layers of uncalcified and calcified cartilage (Figure 4). More superficially, the uncalcified region may be divided into three zones: Superficial (tangential), transitional (or intermediate / middle), and deep (or radial). The superficial zone contains thin, elongated chondrocytes and collagen fibrils that parallel the articular surface. The primary function of this layer is tensile strength. An acellular clear film composed of collagen fibrils, the lamina splendens, is the articulating surface of the superficial zone visible upon gross or arthroscopic inspection. Given its proximity to the joint surface, the water content in the superficial zone is not surprisingly the highest amongst the layers (~80%). Proteoglycan content is lowest in this zone.

The transitional zone occupies approximately 50% of the thickness of uncalcified cartilage. This intermediate layer demonstrates thicker, more obliquely oriented collagen fibers. Compared to the superficial zone, the transitional zone has less water and collagen and greater proteoglycan content. Further, chondrocytes in this zone are more round with higher metabolic activity, evidenced by increasing numbers of intracellular organelles like mitochondria, endoplasmic reticulum, and Golgi membranes(Scott 2005).

The deep zone has the lowest water content (65%) of the uncalcified cartilage layers, reflecting its distance from the articular surface. Although the collagen content is lowest, the fiber diameter is greatest in this zone. The fibers are oriented perpendicular to the joint surface, anchoring the uncalcified cartilage layers to the calcified cartilage zone beneath across the undulating tidemark, the threshold of vascular penetration of the underlying subchondral bone. Proteoglycan content is highest in the deep zone. Chondrocytes are round and arranged in vertical columns.

The calcified cartilage zone is a vascularized layer deep to the tidemark. This zone has a high calcium mineral content and low proteoglycan content. Although most of the collagen in articular cartilage is Type II (90% – 95%), there is a small amount of Type X collagen found in this zone, as it is associated with hypertrophic chondrocytes and calcification of cartilage. Beneath the calcified cartilage layer, separated by a thin cement line, is the subchondral bone, consisting of a lamellar cortical bony endplate and underlying cancellous trabeculae(Madry, van Dijk et al. 2010).

Chondrocytes produce the entirety of the content of the ECM, including proteoglycans, collagen, and non-collagenous proteins. Although proteoglycans represent only approximately 10% of the dry weight of articular cartilage, they give it most of its compressive strength(Ulrich-Vinther, Maloney et al. 2003). Glycosaminoglycans (GAGs), chondroitin sulfate (CS) and keratan sulfate (KS) bind to core protein which, in turn, binds to hyaluronic acid (HA) via link protein, forming an aggrecan proteoglycan molecule (Figure 5a). The negative charge associated with GAGs in aggrecan attracts water, thus attempting to increase tissue swelling. However, the collagen fiber network interconnections prevent swelling and tissue pressure increases (Figure 5b). This property is unique and gives articular cartilage its resilience to compression and deformation.

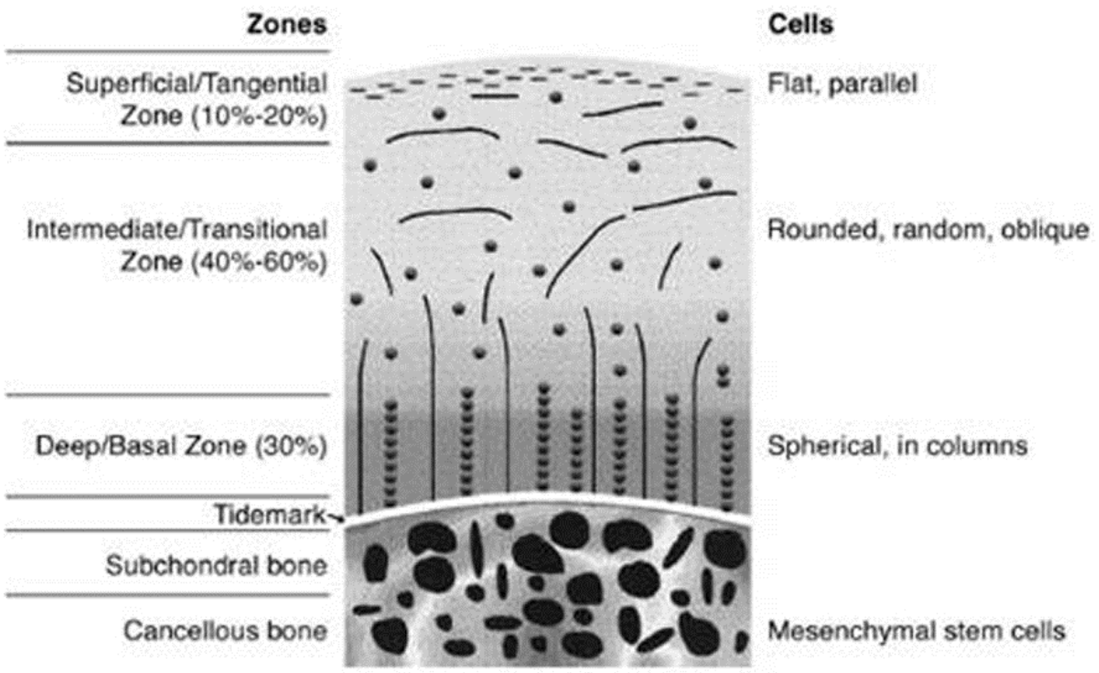

Fig. 4. 4) Schematic depiction of chondrocyte and collagen fibril distribution within the layers of articular cartilage (reproduced with permission from Ulrich-Vinther M, et al: Articular cartilage biology, in *Journal of the American Academy of Orthopaedic Surgeons* 2003; 11: 422. Publisher AAOS)

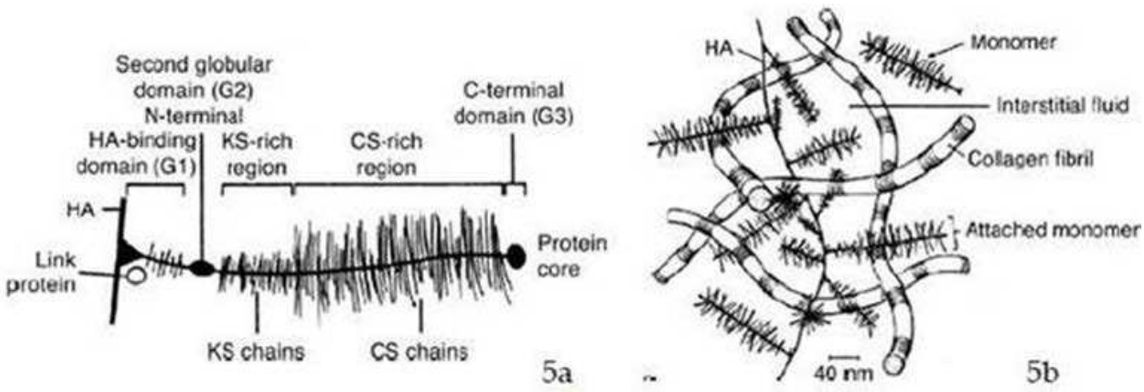

Fig. 5. 5a) Proteoglycan aggrecan molecule composed of chondroitin (CS) and keratan sulfate (KS) glycosaminoglycans, a protein core, and link protein attached to hyaluronic acid (HA) chain; 5b) ECM structure of collagen fibrils intertwined in aggrecan molecules (reproduced with permission from Ulrich-Vinther M, et al: Articular cartilage biology, in *Journal of the American Academy of Orthopaedic Surgeons* 2003; 11: 423. Publisher AAOS)

3. Focal articular cartilage injury

3.1 Prevalence and natural history

Chondral defects in the knee may be seen in up to 63% of knee arthroscopies(Curl, Krome et al. 1997). The prevalence of arthroscopically-detected full-thickness defects is 16%(Flanigan, Harris et al. 2010). Full-thickness focal lesions with an area of 1 cm^2 to 2 cm^2 are seen in approximately 5% of all knee arthroscopies in patients less than 40 years of age(Hjelle, Solheim et al. 2002; Aroen, Loken et al. 2004; Widuchowski, Widuchowski et al. 2007). In an exclusively athletic population, chondral pathology is more common than in the general population. The overall prevalence of full-thickness defects in this population is 36%(Flanigan, Harris et al. 2010). Further, the prevalence of full-thickness lesions is 59% in an asymptomatic group of professional basketball players and runners. The reasons for the increased prevalence in the athlete are multifactorial. Compared with the general population, athletes are 12 times more likely to develop osteoarthritis of the knee(Roos 1998; Drawer and Fuller 2001).

The natural history of the isolated chondral defect and to what degree the isolated defect may become symptomatic is incompletely understood(Buckwalter 1998). Full-thickness lesions may progress due to biomechanical overload with stress concentration around the rim of a defect(Guettler, Demetropoulos et al. 2004), subchondral bone structural changes(Minas and Nehrer 1997), and intra-articular inflammatory cytokine concentration elevations(Fraser, Fearon et al. 2003). Full-thickness defects obviate the shock-absorbing and load-transmitting function of articular cartilage(Minas 1999). The subchondral bone eventually bears the load (Figure 6). Subchondral bone overgrowth has been observed in patients undergoing autologous chondrocyte implantation (ACI), especially in more chronic, larger defects on the lateral femoral condyle(Henderson and LaValette 2005). The subchondral plate becomes sclerotic with vascular congestion and periarteriolar nociceptive fiber stimulation(Minas, Gomoll et al. 2009). The stiffer subchondral plate alters the biomechanical properties of the subchondral bone-articular cartilage interface, which increases shear forces with weight-bearing. Further, subchondral plate thickening and sclerosis due to tidemark advancement is a component of osteoarthritis(Radin and Rose

1986; Burr and Radin 2003). With increasing defect size, these osteocartilaginous changes can only be more greatly accelerated(Flanigan, Harris et al. 2010).

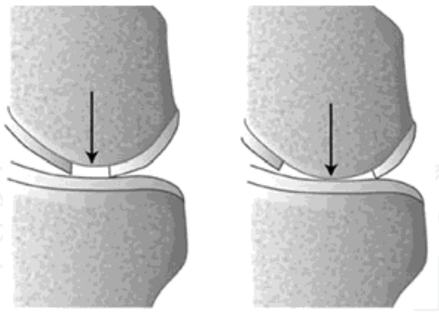

Fig. 6. Well-shouldered, small full-thickness chondral defect with no contact on underlying subchondral bone (left); larger full-thickness defect exhibits subchondral bone contact by the opposing surface (reproduced with permission from The American Academy of Orthopaedic Surgeons in: Jones D and Peterson L: Autologous chondrocyte implantation, Lecture in *Journal of Bone and Joint Surgery, American* 2006; 88A(11): 2503. Publisher AAOS)

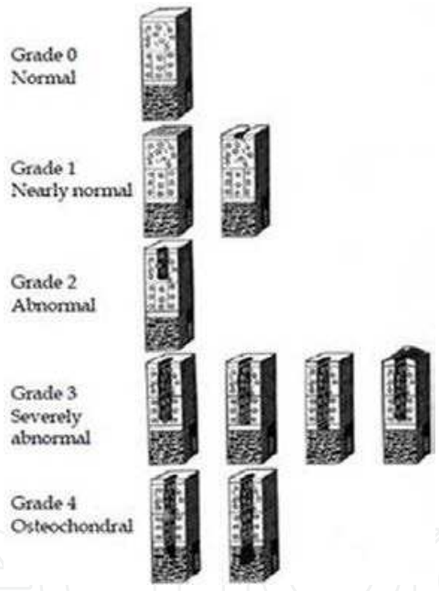

Fig. 7. International Cartilage Repair Society (ICRS) cartilage injury classification (reproduced from the ICRS Cartilage Injury Evaluation Package [www.cartilage.org], with permission from the ICRS).

3.2 Classification systems

The two most commonly used classification systems for arthroscopic analysis of chondral defects in the knee are the Outerbridge system and the International Cartilage Repair Society (ICRS) system (Figure 7). The Outerbridge system grades defects I – IV(Outerbridge 1961). Grade 1 lesions exhibited softening or swelling of cartilage; Grades 2 and 3 both exhibit fragmentation and fissuring of cartilage, with Grade 2 being less than ½ inch and Grade 3 being greater than ½ inch diameter; Grade 4 defects exhibit subchondral bone exposure. The newer ICRS system(Brittberg and Winalski 2003) is advantageous as it

accounts not only for lesion area (cm^2), but also depth, while Outerbridge does not. Osteochondritis dissecans lesions can also be classified according to a similar ICRS-OCD system(Brittberg and Winalski 2003). This classification is based on lesion stability.

3.3 Clinical presentation

Patients with focal chondral defects of the knee may be asymptomatic. Articular cartilage is an aneural tissue. Thus, the presence of a defect does not necessarily produce pain. However, patients with full-thickness chondral defects may demonstrate major limitations in pain and function, according to the Knee Injury and Osteoarthritis Outcomes Score (KOOS)(Heir, Nerhus et al. 2010). In fact, the KOOS quality of life subscore for patients with focal cartilage defects were not significantly different from those patients with OA enrolled for knee osteotomy or arthroplasty. Further, patients with cartilage defects had significantly worse overall KOOS and all KOOS subscores versus patients with anterior cruciate ligament (ACL) deficiency. Patients with chondral defects may also have other concurrent extra- and intra-articular confounders, which make the diagnosis of chondral pathology difficult. Nevertheless, patients with symptomatic chondral defects generally complain of activity-related pain located in a region that correlates with the intra-articular location of the defect for tibiofemoral defects. Patellofemoral lesions generally cause anterior knee pain, worse with prolonged knee flexion or stair climbing. The exact mechanism to account for pain due to pathology in an aneural tissue is not completely understood. However, stimulation of nociceptive fibers in the subchondral bone is one current accepted theory(Mach, Rogers et al. 2002). Further, inflammatory cartilage breakdown products may cause joint effusion with capsular distension in conjunction with synovitis, both leading to joint pain. Patients with chondral flaps may also present with mechanical symptoms such as catching or clicking. Clearly, diagnosis of chondral pathology is complex and requires a thorough history and physical examination, with imaging and arthroscopic examination often required.

4. Articular cartilage imaging

Advances in technology have allowed improved imaging of articular cartilage. These include more sensitive and specific magnetic resonance imaging (MRI) sequences. Further, the ability to both directly and indirectly analyze cartilage qualitatively and quantitatively and its biochemical composition has been enhanced with MRI techniques like dGEMRIC (delayed gadolinium-enhanced MRI of cartilage), T1-rho, T2 mapping, sodium imaging, and diffusion-weighted imaging.

4.1 X-ray

Standard radiographs should always be included in the workup of articular cartilage pathology. Generally speaking, the presence of diffuse arthritic change precludes most cartilage repair or restoration procedures. The 45 degree weight-bearing posteroanterior x-ray (Rosenberg view) (Figure 8) is the most accurate, sensitive and specific for detection of major degenerative changes in the tibiofemoral joint(Rosenberg, Paulos et al. 1988). However, x-ray is extremely important in analysis of the weight-bearing mechanical axis (Figure 8) of the lower extremity for those patients that are enrolled for cartilage surgery.

Articular cartilage defects in the medial compartment are at higher risk for progression if varus malalignment exists (and lateral compartment for valgus, as well)(Linden 1977; Hughston, Hergenroeder et al. 1984; Messner and Maletius 1996; Sharma, Song et al. 2001).

Therefore, surgical correction of tibiofemoral malalignment to neutral or overcorrection is recommended in conjunction with most cartilage surgery (Figure 8). Thus, in addition to standard radiographic workup (extension standing anteroposterior [AP], Rosenberg view, lateral, and Mercer Merchant views), the full-length bilateral hip-to-ankle x-ray allows calculation of mechanical axis of the limb and the necessary alignment correction.

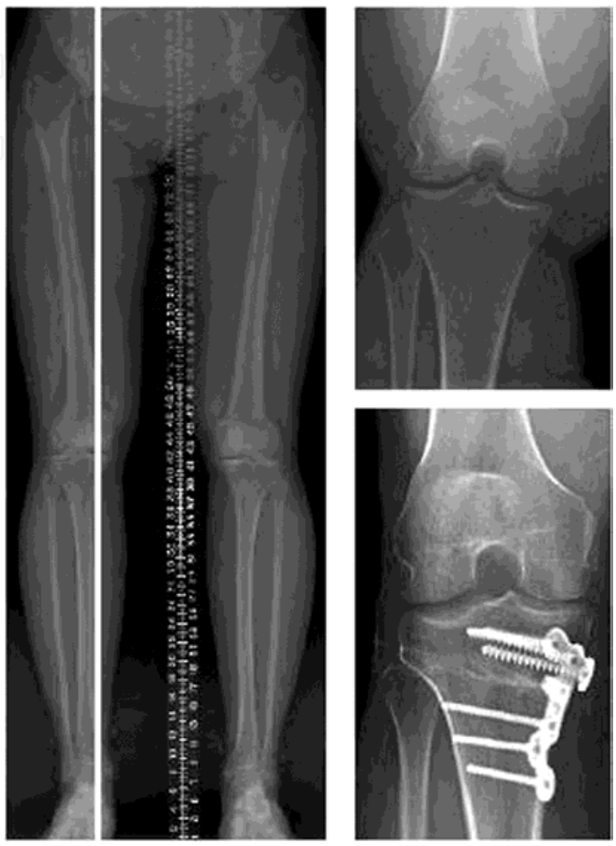

Fig. 8. Left) Standing hip-to-ankle x-ray demonstrating mechanical axis of lower extremity (in medial compartment); Right upper) Rosenberg view (no evidence of OA); Right lower) Rosenberg view after high tibial osteotomy.

4.2 Magnetic resonance imaging (MRI)

MRI is highly advantageous in imaging articular cartilage. This non-invasive modality avoids ionizing radiation, has superior sensitivity and specificity for articular cartilage, and allows for high contrast with proximate structures. Standard MRI sequences in imaging cartilage include conventional spin-echo (SE) and gradient-recalled echo (GRE), and fast SE sequences. The morphologic features of cartilage, evaluated with these standard techniques, can be semi-quantitatively analyzed with the WORMS (whole-organ MRI score)(Peterfy, Guermazi et al. 2004). Also, the MOCART (magnetic resonance observation of cartilage repair tissue) has been demonstrated to be accurate, reliable, and reproducible in post-ACI assessment of cartilage restoration tissue(Marlovits, Singer et al. 2006). Fast SE sequences are included in the ICRS cartilage repair evaluation package for non-invasive assessment of cartilage following surgery. Fat-suppression techniques increase the contrast between articular cartilage and the

underlying subchondral bone. Short-tau inversion recovery (STIR) sequences are an example of a fat-suppression technique used for imaging cartilage defects.

T1-weighted series illustrate anatomic features of articular cartilage well, but have poor contrast between it and synovial fluid. T2-weighted series demonstrate better contrast between cartilage and joint fluid. Proton-density-weighted series are an intermediate, providing high contrast and excellent intra-cartilaginous structure.

The biphasic extracellular matrix of articular cartilage includes both fluid and a collagen-aggrecan network. The negatively-charged GAGs of aggrecan molecules allow for ions like gadolinium diethylenetriaminepentaacetic acid $(Gd\text{-}DTPA)^{-2}$ and sodium (Na^{+}) to interact to quantitatively measure proteoglycan content(Crema, Roemer et al. 2011). dGEMRIC utilizes $Gd\text{-}DTPA^{-2}$ based on the fact that its negative charge allows it to cluster where the GAG content is relatively low. Thus, since $Gd\text{-}DTPA^{-2}$ concentration is measured via T1, T1 mapping after intravenous (IV) $Gd\text{-}DTPA^{-2}$ allows for quantitative assessment of GAG concentration(Crema, Roemer et al. 2011). Higher $Gd\text{-}DTPA^{-2}$ indicates lower GAG content, while lower $Gd\text{-}DTPA^{-2}$ indicates higher GAG content.

While standard T2-weighted series provide a qualitative assessment of extracellular matrix of cartilage, T2 mapping quantitatively describes variations in relaxation time of cartilage via collagen network-water interaction. Higher T2 is seen in early stages of degenerative osteoarthritis(Dunn, Lu et al. 2004). Although T2 maps do not demonstrate any relationship between T2 and grade of defect or arthritic change (marker of severity of disease)(Koff, Amrami et al. 2007), they have demonstrated the ability to longitudinally assess cartilage repair or restoration tissue following surgery(Welsch, Mamisch et al. 2008).

Just as dGEMRIC may measure proteoglycan content, T1-rho values can also be used to assess extracellular matrix composition. The loss of articular cartilage early in osteoarthritis displays a higher T1-rho value than normal cartilage(Stahl, Luke et al. 2009). T1-rho measures not only proteoglycan content, but also collagen and other non-collagen proteins within the matrix(Mlynarik, Trattnig et al. 1999). This increased sensitivity makes it useful for detection of early arthritic change.

Sodium is a positively-charged ion that must equilibrate exactly with the negative charge imparted by GAGs in ECM of articular cartilage. Thus, normal hyaline articular cartilage exhibits high sodium content, while chondral defects and osteoarthritis exhibit lower sodium content due to loss of GAGs. This makes sodium imaging techniques attractive due to its ability to directly measure GAG content without the use of contrast material.

5. Articular cartilage surgery

Articular cartilage surgery can be broadly grouped into three categories: Palliative techniques that are, as the name implies, intended to relieve pain secondary to chondral pathology; repair techniques that invoke stimulation of the underlying subchondral bone marrow (MST), including microfracture, subchondral drilling, and abrasion arthroplasty; and restoration techniques that attempt to transfer or produce normal hyaline articular cartilage, including autologous chondrocyte implantation (ACI), osteochondral autograft / mosaicplasty, osteochondral allograft, and other cell-based surgical treatments.

5.1 Palliative techniques

Palliative techniques are minimally-invasive, arthroscopic surgeries intended to relieve pain due to articular cartilage disease. Debridement consists of removal of unstable, loose flaps or

fronds of articular cartilage and loose bodies. This heterogeneous definition also encompasses lavage, which removes inflammatory joint fluid containing catabolic enzymes. All potentially mechanically-irritating pathology is removed and unstable, irregular edges of articular cartilage and meniscal tissue are smoothed.

Arthroscopic debridement may be indicated in certain groups of patients. The American Academy of Orthopaedic Surgeons (AAOS) Clinical Practice Guidelines (Level V evidence; Grade of Recommendation C)(Richmond, Hunter et al. 2009) recommend arthroscopic partial meniscectomy and / or loose body removal in patients with symptomatic osteoarthritis with primary complaints (mechanical symptoms) of torn meniscus or loose body. However, these guidelines do not recommend arthroscopic debridement or lavage in patients with symptomatic osteoarthritis without mechanical symptoms (Level I and II evidence; Grade of Recommendation A).

In patients with isolated chondral defects, the post-operative rehabilitation following certain cartilage repair or restoration techniques may preclude their use. Some athletes (professional or amateur) may not be willing to forego part of a competitive season or at least one full season due to concerns of scholarships, salaries, contracts, signing bonuses, other endorsements, public image, and career length. Further, many athletes are aware of certain surgical techniques and media coverage has instilled preconceived, irrational notions about their efficacy. Thus, arthroscopic debridement may be an effective, quick method to return an athlete to sport. Do not perform microfracture or other more advanced cartilage surgery if the patient has not consented or is unwilling to undergo the rehabilitation following surgery.

5.1.1 Surgical technique

Standard arthroscopic portals (working anteromedial and viewing anterolateral) are generally all that are required for this technique. Systematic diagnostic arthroscopy ensures that each location in the joint is inspected and no pathology left untreated. This includes the suprapatellar pouch, medial and lateral gutters, menisci, chondral surfaces, and cruciate ligaments within the notch. If access to the posterior compartments is indicated, the arthroscope may be placed through the notch and accessory posteromedial or posterolateral portals created to evaluate for loose bodies or posterior horn meniscal pathology. Some loose bodies may be removed with suction on an arthroscopic shaver, however others may require a grasper and either a separate incision or enlarging one of the standard portals. Degenerative chondral flaps and meniscal tears may be removed or trimmed to stable, smooth edges with a combination of arthroscopic biters, shavers, and curettes. Thorough palpation of all surfaces with a probe ensures no pathology is missed. If osteophytes are present, an arthroscopic burr may be required to contour this down as it may be a source of mechanical impingement and loss of motion.

5.1.2 Outcomes

Short- and mid-term outcomes of arthroscopic debridement are good to excellent (variably defined) in up to 75% of patients(Sprague 1981; Fond, Rodin et al. 2002). Patients whose primary symptom is mechanical generally have a better prognosis(Baumgaertner, Cannon et al. 1990; Ogilvie-Harris and Fitsialos 1991). Shorter duration of symptoms(Yang and Nisonson 1995; Fond, Rodin et al. 2002), normal coronal plane alignment(Baumgaertner, Cannon et al. 1990; Aaron, Skolnick et al. 2006), and no evidence of joint space narrowing(Jackson and Dieterichs 2003; Aaron, Skolnick et al. 2006) also are predictive of better outcomes.

5.2 Cartilage repair techniques

Cartilage repair techniques intend to stimulate the subchondral bone marrow (marrow-stimulation techniques, MST) to induce mesenchymal stem cell infiltration into a chondral defect with formation of a clot that may differentiate into repair tissue. This tissue is generally fibrocartilage, with a ratio of Type II to I collagen that is less than that of normal hyaline articular cartilage. The biomechanical properties and durability of fibrocartilage are inferior to that of hyaline cartilage. Microfracture, subchondral bone drilling, and abrasion arthroplasty are MSTs.

5.2.1 Surgical technique

Standard diagnostic arthroscopy is performed prior to assessment of a lesion amenable to microfracture. The use of a tourniquet is not recommended as this precludes assessment of depth of penetration of arthroscopic awl with egress of marrow fat or blood. The defect is prepared by creation of vertical walls with stable rims with a full radius resector or curette and removal of the calcified cartilage zone with a curette. Thus, poorly-shouldered lesions are not well-suited for this treatment. A stable subchondral plate is desired, so caution is warranted when debriding the calcified cartilage zone so that the plate is not compromised. Arthroscopic awls of variable angles (0°, 30°, 45°, 60°, and 90°) may be used to create multiple holes, the microfractures, perpendicular to the surface penetrated. The sequence of hole creation should be centripetal, from the periphery inward, approximately 3-4 mm apart and 3-4 mm deep (Figure 9). Do not place holes so close as to converge upon one another. Once complete, reduce arthroscopic pump pressure to visualize marrow contents from each of the holes. Do not use an intra-articular drain post-operatively, as this will remove the desired clot formation within the defect. Steadman has stressed the importance of post-operative rehabilitation following microfracture. Immediate continuous passive motion (CPM) is indicated for at least 8 hours per day for at least 8 weeks. Return to competitive sports is not allowed prior to 6 to 9 months.

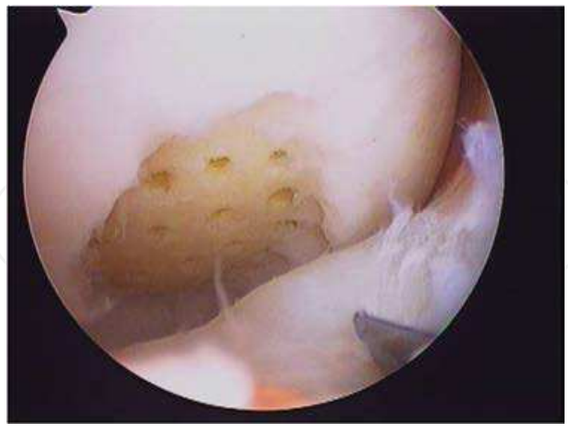

Fig. 9. Arthroscopic photograph of microfracture of medial femoral condyle defect.

5.2.2 Outcomes

In horse and human studies, microfracture has been consistently shown to produce a greater quantity of repair tissue versus no treatment of an isolated chondral defect(Frisbie, Trotter et al. 1999; Mithoefer, McAdams et al. 2009). Better outcomes have been demonstrated with the

creation of smooth, vertical walls of the defect and removal of the calcified cartilage layer directly beneath the tidemark(Frisbie, Morisset et al. 2006).
Microfracture is generally indicated for a full-thickness, chondral defect (after debridement of the defect to stable rims with exposed bone) of the femoral condyles, trochlea, patella, or tibial plateau. The pioneer of microfracture (Steadman) has successfully utilized microfracture in degenerative arthritis(Miller, Steadman et al. 2004). Although microfracture has been used in high-performance athletes (NFL) with excellent outcomes and return-to-play(Steadman, Miller et al. 2003), other studies have shown less success in high-performance professional athletes, with low rates of return-to-sport and decreased performance if able to return(Cerynik, Lewullis et al. 2009; Namdari, Baldwin et al. 2009).
Outcomes of microfracture are mixed. Long-term outcomes have been successful (mean 11 years, range 7 to 17 years) in patients less than 45 years age, without malalignment or meniscal or ligamentous pathology, graded by both subjective and objective outcome measures(Steadman, Briggs et al. 2003). Recent systematic reviews have shown excellent short-term clinical outcomes following microfracture(Mithoefer, McAdams et al. 2009; Harris, Siston et al. 2010). However, after 18 to 24 months, outcomes tend to deteriorate, especially in patients with defects larger than 2 to 4 cm^2, longer pre-operative duration of symptoms, prior surgeries to the knee, and older age (Mithoefer, McAdams et al. 2009; Harris, Brophy et al. 2010; Harris, Siston et al. 2010). Further, microfracture may compromise future outcomes following ACI. A three times greater risk of failure after ACI has been shown in those patients with previous microfracture versus those without(Minas, Gomoll et al. 2009). In general, it appears that microfracture is best suited for younger patients with small defects who have normal alignment and a short pre-operative duration of symptoms and are willing to comply with post-operative rehabilitation.

5.3 Cartilage restoration techniques

Cartilage restoration techniques either transfer (mosaicplasty, osteochondral autograft and allograft) or attempt to produce (cell-based treatments such as ACI) normal hyaline articular cartilage.

5.3.1 Osteochondral autograft / mosaicplasty

Osteochondral autograft (OAT) and mosaicplasty are two similar techniques that harvest an osteochondral plug(s) from a "less weight-bearing" part of the knee and transplant them to a defect on a more weight-bearing, articulating location. Given the three-dimensional complexity of the articular surfaces of the knee, one can anticipate that stable congruity of the transplanted plug is paramount to the procedure's technical success. This procedure (Figure 10) can place one or many plugs of variable sizes to fill a defect. If one plug is used and is flush with surrounding cartilage, no fibrocartilaginous tissue from the underlying subchondral bone will be formed. If more than one plug is used, however, the intervening areas fill with fibrocartilage. Since this is an osteochondral transplant, chondral *and* osteochondral defects may be treated without the need for bone grafting (as opposed to other cartilage surgery). This technique, however, is limited by donor-site supply. This has prompted most authors to limit the size transplanted to no greater than 4 or 5 cm^2. Despite concerns for donor-site morbidity following harvest, long-term donor-site complaints (measured by Bandi score) are minor and present in small numbers of patients, including high-level athletes (3% - 5%)(Hangody, Vasarhelyi et al. 2008; Hangody, Dobos et al. 2010).

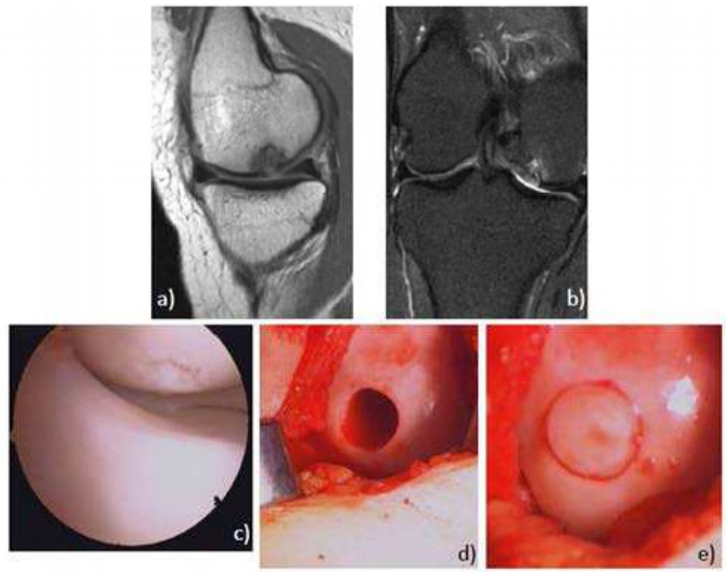

Fig. 10. 10a) Sagittal MRI demonstrating osteochondral defect of medial femoral condyle; 10b) Coronal MRI demonstrating same defect; 10c) Arthroscopic photograph of defect; 10d) Mini-arthrotomy image after recipient site preparation; 10e) Flush plug placed.

5.3.1.1 Surgical technique

Osteochondral autograft may be performed all-arthroscopically or via mini-arthrotomy. Either is acceptable, although arthroscopically may be technically more demanding. Standard diagnostic arthroscopy is performed initially. The defect is prepared by obtaining stable smooth edges with vertical walls. Once this is complete, the defect is then precisely measured and templated. There are several unique proprietary designs available to harvest and place plugs. However, the general principles of each are the same: A sharp cutting harvester, perpendicular to the surface, is impacted to a pre-determined depth and donor plug is harvested. The size of the harvester can be range from 2.5 to 10 mm in diameter. Donor sites include the intercondylar notch, and superomedial and superolateral borders of the femoral condyles. These donor sites should properly be described as "less weight-bearing" regions, rather than non-weight-bearing regions(Simonian, Sussmann et al. 1998; Ahmad, Cohen et al. 2001). The recipient site is prepared to accept the graft to the correct depth. The plug is then placed press-fit via instrumented manual impaction. Plugs should be placed flush circumferentially, as plugs placed proud demonstrate significantly greater contact pressure around the plug's rim and the opposing surface(Harris, Solak et al. 2011). Even plugs countersunk beneath the surrounding normal cartilage create significant pressure increases around the rim of the normal cartilage(Koh, Wirsing et al. 2004). It cannot be overemphasize that critical for this procedure's success is flush plug placement achieved via perpendicular plug harvest and transplantation.

5.3.1.2 Outcomes

Outcomes following osteochondral autograft / mosaicplasty have been largely good or excellent. In an exclusively athletic population of nearly 400 patients at nearly 10 year mean

follow-up, good or excellent clinical outcomes were observed in 91% of femoral condyle OAT, 86% tibial, and 74% patellofemoral(Hangody, Dobos et al. 2010). The timing of return to sport after OAT is faster than that after microfracture or ACI(Harris, Brophy et al. 2010). Also, the rate of return to sport and overall clinical outcomes after OAT are better than that after microfracture(Harris, Brophy et al. 2010). Except for more rapid clinical improvement, no significant difference has been demonstrated between OAT and ACI with regard to clinical outcomes(Harris, Brophy et al. 2010; Harris, Siston et al. 2010).

5.3.2 Osteochondral allograft

Principles of osteochondral allograft are similar to those of autograft, with the difference being the source of the osteochondral plug. Although concern for disease transmission, cell viability, and host-graft immunogenicity exist, this technique is a very useful treatment for larger chondral and osteochondral defects (usually greater than 2 to 4 cm^2). There is no limitation to the size of graft used, as entire condyles may be transplanted. Given the size constraints imposed by the transplanted graft, most allografts are implanted via an arthrotomy, although some cases may allow all-arthroscopic placement, just as with OAT.

5.3.2.1 Surgical technique

Just as with OAT, the defect is prepared to stable smooth rims with vertical walls using a sharp curette or full-radius resector and then sized. The cylindrical dowel graft is then prepared to match the size of the defect. The dowel graft is then press-fit into its recipient socket via instrumented manual impaction. Supplemental fixation is generally not required. A shell graft technique is another viable option when the dowel technique is not possible because of defect location or size. The shell is prepared freehand and usually requires fixation. This technique is technically more demanding than the dowel.

5.3.2.2 Outcomes

Outcomes after osteochondral allograft demonstrate good to excellent outcomes in 72% to 94% of patients at long-term follow-up with 5 year Kaplan-Meier survivorship around 95%, 10 year survival around 80% - 85%, and 15 year survival around 65%(Garrett 1994; Shasha, Krywulak et al. 2003; Gross, Shasha et al. 2005; Emmerson, Gortz et al. 2007). Although technically demanding, osteochondral allograft has long-term proven success in patients with larger defects and bone loss that may have failed a prior cartilage surgery.

5.3.3 Autologous chondrocyte implantation (ACI)

ACI is a two-stage cartilage restoration technique indicated for lesions greater than 2 cm^2 on the femoral condyles, trochlea, or patella. Stage 1 involves arthroscopic assessment of the defect and a full-thickness cartilage biopsy. Stage 2 involves cell implantation via arthrotomy under a periosteal or collagen membrane patch or, more recently, outside the U.S., cell placement onto a three-dimensional scaffold that can potentially be placed all-arthroscopically. The premise behind ACI is that a biopsy and growth in culture of your own cells should theoretically produce normal hyaline articular cartilage upon implantation. However, dedifferentiation of chondrocytes when grown in monolayer culture and subsequent re-differentiation upon implantation has produced “hyaline-like” cartilage. This tissue has a Type II collagen and proteoglycan composition that is close, but not identical to that of normal hyaline articular cartilage.

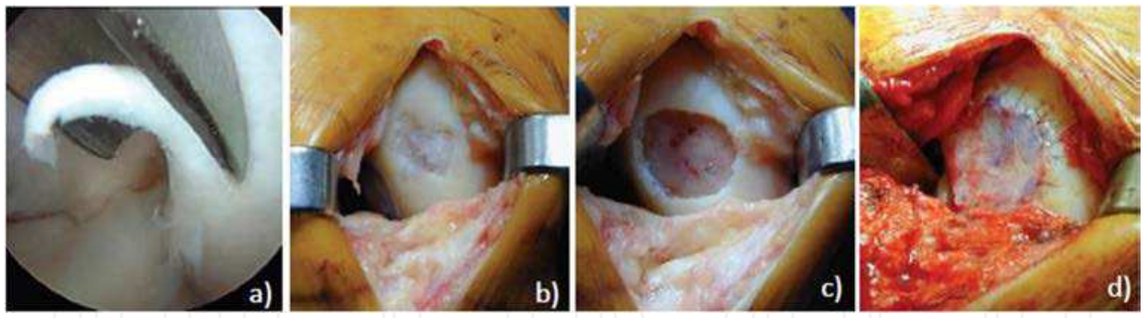

Fig. 11. 11a) Arthroscopic biopsy taken from intercondylar notch; 11b) Knee arthrotomy revealing full-thickness femoral condylar defect; 11c) Defect following debridement to stable rims; 11d) Following suture of periosteal patch and implantation of cultured autologous chondrocytes. (Reproduced with permission from Alford JW and Cole BJ: Cartilage restoration, Part 2: Techniques, outcomes, and future directions, in *American Journal of Sports Medicine* 2005; 33: 443-460. Publisher Sage Publications).

5.3.3.1 Surgical technique

ACI Stage 1 involves standard diagnostic arthroscopy with debridement of the defect and a full-thickness cartilage biopsy taken from the intercondylar notch (Figure 11a), or superomedial or superolateral edge of the medial or lateral femoral condyles, respectively. An arthroscopic ring curette or notchplasty gouge may be used to obtain two or three slivers of cartilage ~ 5 mm x 8 mm (~200 – 300 mg; 200,000 – 300,000 cells). Larger defects may warrant larger amount of tissue. The biopsy should contain a small sample of bone. Currently, the biopsy remains viable for implantation for two years after harvest. It is critical to determine the complete extent and size of the lesion by removing all loose, unstable, undermined, and unhealthy cartilage to well-shouldered, vertical walls. Healthy stable cartilage is required at the time of Stage 2. Upon implantation during Stage 2, removal of all cartilage in the bed is required down to, but not into, the subchondral bone. Any inadvertent penetration into the bone will generate undesirable bleeding. Epinephrine-soaked neuropatties may be used to achieve adequate hemostasis. With stable vertical walls (Figure 11c), the patch can be sutured (using lubricated 6-0 Vicryl suture on a P1 cutting needle) (Figure 11d) in with one small opening remaining at the most superior portion of the patch to allow for cell implantation. Sutures should be spaced 3 to 4 mm apart with a 3 mm bite onto normal cartilage and the knots tied on the patch side. Inject sterile saline under patch to test watertightness prior to inserting cell solution via 18-gauge angiocatheter. Apply fibrin glue as necessary to ensure watertight closure. Once cells are implanted, suture the remaining opening and fibrin glue as needed.

5.3.3.2 Variations in technique (ACI generations)

Currently, three generations of ACI exist. First-generation techniques involve cell implantation under a periosteal or collagen membrane patch via arthrotomy. Second-generation techniques utilize either arthrotomy or arthroscopy to implant cells via cell-seeded, three-dimensional bioabsorbable scaffolds. Third-generation technique uses either arthrotomy or arthroscopy to deliver in-vitro treated cells within chondro-inductive and chondro-conductive, three-dimensional matrices. Although clinical outcomes of first- and second-generation ACI are not significantly different, first-generation (especially with periosteal cover) has a significantly greater number of complications, failures, and unplanned re-operations than second-generation(Harris, Siston et al. 2011).

5.3.3.3 Outcomes

Outcomes after ACI are good to excellent in approximately 90% of patients at short- and mid-term follow-up(Peterson, Minas et al. 2000; Bentley, Biant et al. 2003; Mandelbaum, Browne et al. 2007). Long-term follow-up reveals 92% patient satisfaction with significant improvements in subjective and objective clinical outcome scores (224 patients at mean 13 year follow-up, range 12 to 20 years)(Peterson, Vasiliadis et al. 2010). Several recent systematic reviews have compared ACI to other cartilage surgeries and have indicated a trend toward improved clinical and tissue outcomes following ACI versus microfracture and OAT at mid- and short-term follow-up(Harris, Siston et al. 2010; Vavken and Samartzis 2010). The quality of evidence in the literature is methodologically poor(Jakobsen, Engebretsen et al. 2005; Harris, Siston et al. 2011) and only further higher quality randomized comparative clinical trials will be able to determine if one cartilage repair or restoration technique is superior.

6. Role of alignment in cartilage surgery

6.1 Role of mechanical axis of lower extremity

The mechanical axis of the lower extremity (straight line drawn from center of hip to center of ankle) normally lies just medial to the medial tibial spine. Varus malalignment brings this axis further inside the medial compartment or even medial to the joint. With axial loading, varus malalignment causes increased pressure in the medial compartment cartilage(Loening, James et al. 2000). Increased stress may negatively impact cartilage repair and restoration procedures. Without correction of the alignment to at neutral, the outcomes of cartilage procedures have been less successful in the presence of varus malalignment. This has led to increased performance of valgus-producing high tibial osteotomy (HTO) either via an opening- (OW-HTO) or closing-wedge (CW-HTO) technique. Mechanical axis correction to neutral or slight valgus is adequate in conjunction with cartilage repair or restoration procedures(Mina, Garrett et al. 2008). For medial compartment osteoarthritis, overcorrection to up to 62% of the width of the tibial plateau from the medial tibial border is warranted(Miller, Cole et al. 2008). A similar technique is used for lateral compartment chondral pathology in the setting of valgus malalignment via a laterally-based opening wedge technique.

6.1.1 Outcomes

Outcomes after combined HTO and cartilage surgery for medial compartment cartilage pathology and varus malalignment have demonstrated significant improvements in subjective and objective clinical measures. Both CW-HTO and OW-HTO techniques have seen similar success concurrent with microfracture(Sterett and Steadman 2004; Sterett, Steadman et al. 2010; Pascale, Luraghi et al. 2011), abrasion arthroplasty(Matsunaga, Akizuki et al. 2007), and ACI(Franceschi, Longo et al. 2008; Gomoll, Kang et al. 2009; Minas, Gomoll et al. 2009).

6.2 Role of patellofemoral alignment

Similar to unloading osteotomy and cartilage surgery for tibiofemoral joint articular cartilage lesions with malalignment, patellofemoral joint chondral pathology in the setting of patellofemoral malalignment also warrants unloading via tibial tubercle osteotomy when combined with cartilage surgery. In the setting of lateral patellar or trochlear defects, unloading via osteotomy should include either medialization (Elmslie-Trillat) or

anteromedialization (Fulkerson). The degree of medialization needed may be estimated with pre-operative measurement of the TT-TG (tibial tubercle-trochlear groove) distance. Nevertheless, the surgeon must be cognizant during the pre-operative workup and the operation itself to assure that no medial patellar or trochlear pathology exists if planning to unload the lateral patellofemoral compartments, as this will increase stress on degenerative cartilage(Kuroda, Kambic et al. 2001). Distal patellar cartilage pathology may warrant anteromedialization to allow the patella to enter the trochlea in earlier degrees of flexion and unload the distal cartilage pathology(Colvin and West 2008). In the presence of lateral patellar tilt, a lateral retinacular release may be indicated(Arendt 2009). Medial patellofemoral ligament (MPFL) insufficiency may warrant reconstruction(Arendt 2009).

6.2.1 Outcomes

The clinical outcomes following patellofemoral realignment osteotomy have demonstrated success with the proper indications. In patients with lateral and distal patellar defects, anteromedialization led to 100% patient satisfaction with 87% good to excellent results, while patients with medial, proximal, or diffuse defects had only 43% good to excellent results(Pidoriano, Weinstein et al. 1997). Excellent short- and mid-term outcomes have been demonstrated when distal patellofemoral realignment has been combined with ACI(Bentley, Biant et al. 2003; Minas and Bryant 2005; Henderson and Lavigne 2006; Farr 2007; Gigante, Enea et al. 2009; Gobbi, Kon et al. 2009).

7. Role of meniscus in cartilage surgery

7.1 Post-meniscectomy knee and meniscus allograft transplantation (MAT)

Primary functions of the meniscus include load transmission, shock absorption, and secondary joint stability. When the meniscus is anatomically (post-meniscectomy) or functionally (complete radial tears, root tears) lost, the ipsilateral articular cartilage now wholly bears the compartmental load. Thus, meniscal preservation is key to articular chondroprotection. In the young patient with meniscal deficiency, MAT is a viable treatment option. Key surgical tenets include proper sizing, graft bank source, bony rather than suture fixation, and recognition of associated chondral disease. Historically, ipsilateral full-thickness cartilage pathology was considered a contraindication to MAT due to the poor clinical outcomes seen after MAT in this patient group(Noyes and Barber-Westin 1995; van Arkel and de Boer 1995). Given the recent improvements in outcomes following cartilage surgery and MAT when considered in isolation, the combined procedure (either simultaneous or staged) has received much attention. In the presence of combined meniscal deficiency and advanced chondral pathology, cartilage surgery may be a necessary adjunct to MAT for optimal biological joint preservation.

7.1.1 Outcomes

Outcomes following combined MAT and cartilage surgery (ACI, OAT, osteochondral allograft) have demonstrated equivalent subjective and objective clinical outcomes as either procedure performed in isolation(Harris, Cavo et al. 2011). In these studies, failure rate is low (12%), but the rate of re-operation is high (~50%). Most of the failures (86%) that occur following combined MAT and cartilage surgery were due to failure of the MAT. In addition to consideration of meniscal status, coronal plane alignment must also be accounted for so as to not overload the ipsilateral compartment that receives cartilage surgery and MAT.

8. Articular cartilage defect management algorithm

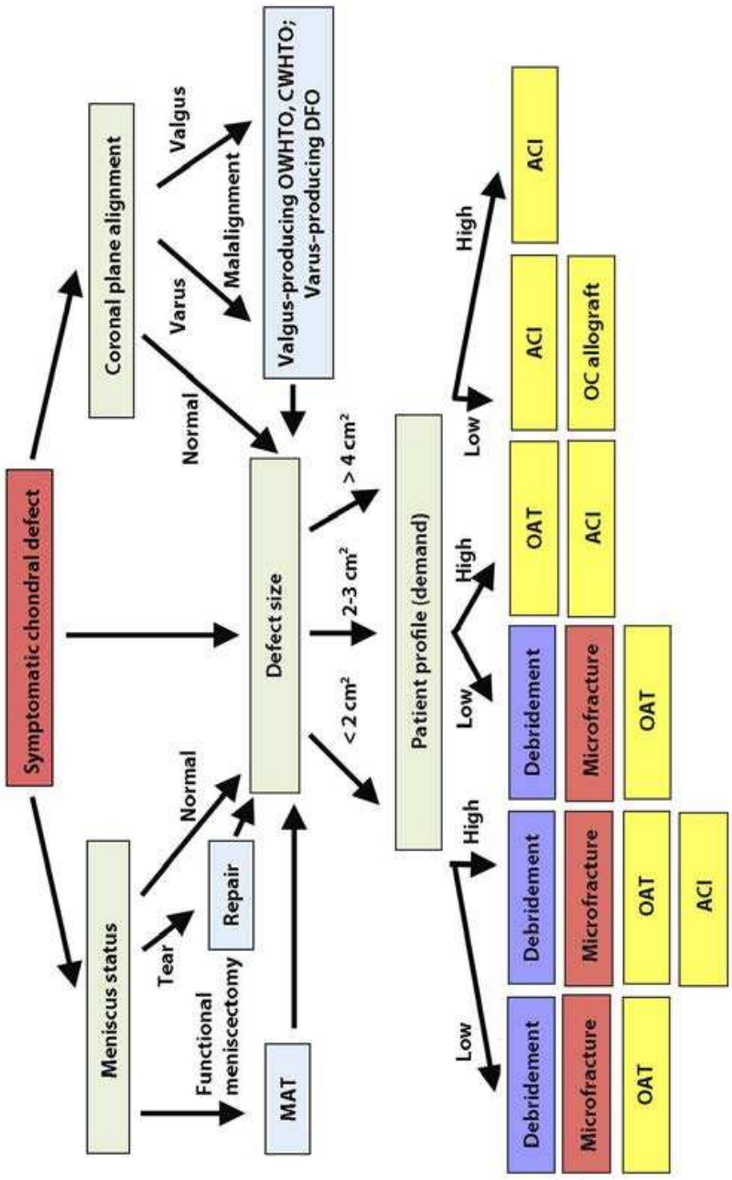

Fig. 12. Management of symptomatic chondral defect of medial or lateral femoral condyle. Concurrent issues, such as meniscal deficiency and coronal plane malalignment, need to be addressed, either simultaneously or sequentially in a staged manner. The most important defect-specific parameter is size (area in cm^2), dictating treatment choice. MAT (meniscus allograft transplantation), HTO (high tibial osteotomy), OWHTO (opening wedge HTO), CWHTO (closing wedge HTO), DFO (distal femoral osteotomy), OAT (osteochondral autograft), ACI (autologous chondrocyte implantation), OC (osteochondral) allograft. Yellow (cartilage restorative technique); Red (cartilage reparative technique); Purple (cartilage palliative technique).

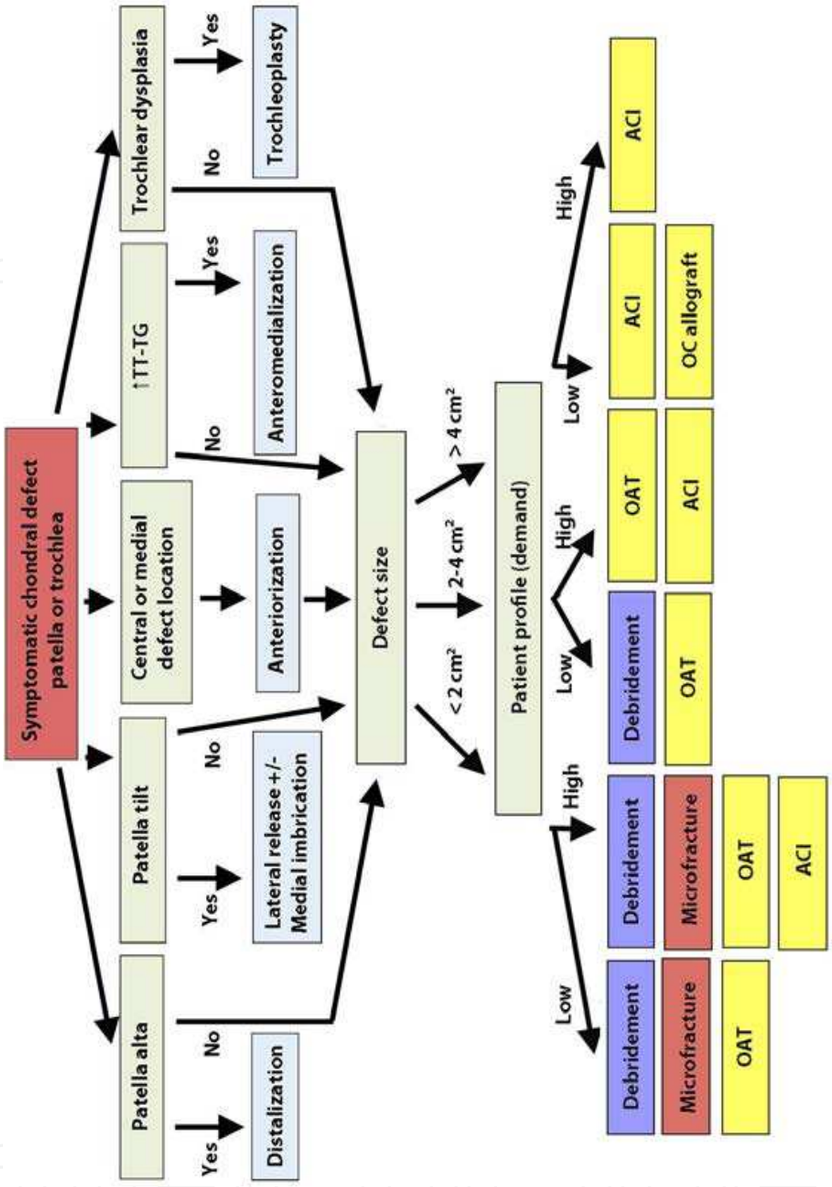

Fig. 13. Management of symptomatic chondral defect of femoral trochlea or patella. Concurrent issues, such as patella alta, patella tilt, increased extensor mechanism lateral vector (tibial tubercle – trochlear groove [TT-TG] distance), and trochlear dysplasia, need to be addressed, either simultaneously or sequentially in a staged manner. Centrally or medially located defects warrant different unloading osteotomy techniques, pending normal alignment. Medial patellofemoral compartment articular cartilage pathology is a contraindication to medializing osteotomy. The most important defect-specific parameter is size (area in cm^2), dictating treatment choice. OAT (osteochondral autograft), ACI (autologous chondrocyte implantation), OC (osteochondral) allograft. Yellow (cartilage restorative technique); Red (cartilage reparative technique); Purple (cartilage palliative technique).

9. Conclusions

Articular cartilage defects of the knee are a common source of pain and disability. Their natural history progression to osteoarthrosis of the knee is not completely understood. The management of these lesions is clearly multifactorial, involving factors specifically related to the patient, the lower extremity, the knee and the defect. Several surgical procedures exist to treat these injuries when non-operative management has failed. These include palliative, reparative, and restorative techniques. Both subjective and objective outcomes demonstrate significant improvement following these procedures. The future of cartilage surgery will need high-quality randomized clinical trials, using minimally-invasive techniques with the goal of obtaining normal hyaline articular cartilage, in the hopes of delay or prevention of defect progression to osteoarthrosis of the knee.

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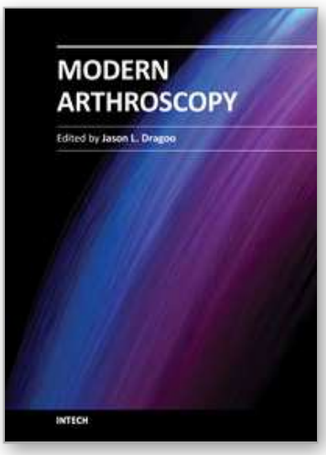

Modern Arthroscopy
Edited by Dr Jason L. Dragoo

ISBN 978-953-307-771-0
Hard cover, 302 pages
Publisher InTech
Published online 09, December, 2011
Published in print edition December, 2011

Modern Arthroscopy will assist practitioners to stay current in the rapidly changing field of arthroscopic surgery. The chapters in this book were written by a panel of international experts in the various disciplines of arthroscopy. The goals of this text are to present the classical techniques and teachings in the fields of Orthopaedics and Dentistry, but also to include new, cutting-edge applications of arthroscopy, such as temporomandibular arthroscopy and extra-articular arthroscopy of the knee, just to name a few. We hope Modern Arthroscopy becomes a core reference for your arthroscopic surgery practice.

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