

Practical MR Imaging of Articular Cartilage

John F. Feller

INTRODUCTION

The development of new therapeutic options for the treatment of articular cartilage pathology and new insights into the pathophysiology of arthritic disorders have fueled interest in developing better means of non-invasively evaluating hyaline articular cartilage. Specifically, the status of articular cartilage influences patient selection for chondroplasty, chondrocyte transplantation, arthroplasty and drug therapy. It is also of prognostic value in patients undergoing meniscal surgery. This presentation will overview the emerging role of MR imaging as an accurate non-invasive technique for the evaluation of hyaline articular cartilage in normal and diseased states. Recent practical MR technical advances will also be discussed including: the use of quadrature and phased array coils; high resolution 512 matrix acquisitions; the value of various pulse sequences; and the relative merits of high field versus low field MR imaging.

CARTILAGE STRUCTURE AND FUNCTION

Articular cartilage provides a nearly frictionless gliding articular surface and is capable of bearing enormous loads while evenly distributing these forces to underlying subchondral bone. Hyaline cartilage is capable of withstanding an enormous load with little deformation and demonstrates high durability typically able to withstand a lifetime of use. A review of the histology of articular cartilage reveals that it is an avascular, alymphatic, and aneural tissue composed predominantly of an extracellular matrix containing a relatively small number of chondrocytes. The extracellular matrix is composed predominantly of water, collagen and proteoglycans. Water is the single largest component constituting 60-80% of the total weight of the extracellular matrix. Collagen is the second largest component constituting approximately 60% of the dry weight. Most of the collagen in articular cartilage is Type II collagen. There is little, if any, Type I collagen present in hyaline cartilage. Type I collagen is the major structural component of fibrocartilage. Proteoglycans constitute approximately 30% of the dry weight of articular cartilage. These proteoglycans contain glycosaminoglycans which are hydrophilic. This creates osmotic, ionic and Donnan forces that result in a net swelling pressure in articular cartilage.

Articular cartilage can be divided into histologic zones on the basis of the orientation of the collagen fibers. The outer zone of articular cartilage is referred to as the tangential or superficial zone and makes up approximately 10% of the thickness of articular cartilage. The collagen fibers within this zone run tangential to the surface of the cartilage. Immediately deep to the superficial zone is the transitional or intermediate zone in which the collagen fibers are obliquely oriented. This zone makes up approximately 40% of the thickness of articular cartilage. The next deeper zone is the radial zone in which the collagen fibers are oriented perpendicular to the cartilage surface. Finally, there is a thin zone of calcified cartilage present at the interface of the articular surface with the underlying subchondral bone. These two deepest zones combine to make up approximately 50% of the articular cartilage thickness.

The orientation of the collagen fibers is important for protection against shear and tensile stresses in articular cartilage. The swelling pressure created by the proteoglycans provides the main protection against compressive loads.

CARTILAGE DAMAGE AND REPAIR

The most common cause of articular cartilage damage is osteoarthritis. This consists of predominantly non-inflammatory degeneration of the articular cartilage in synovial joints. Three stages of osteoarthritis have been proposed. The first stage of this disease process is characterized by disruption of the collagen framework of the articular cartilage, decreases in proteoglycan content and aggregate size and an increase in water content that results in cartilage swelling and softening. The second stage is characterized by a repair response in which the body attempts to repair the damage with increased anabolic and catabolic activity in the cartilage as well as with proliferation of chondrocytes which form clusters or clones of cells. This stage may last several years and can result in increased thickness of articular cartilage. The third and final stage is characterized by inability of the repair mechanisms to keep up with the damage, probably due to decreased cellular proliferation and anabolic activity of the chondrocytes. The result is cartilage loss with fibrillation, fissures, erosion and cracking of the articular cartilage. The response to articular cartilage degeneration includes marginal osteophyte formation, subchondral sclerosis, subchondral cyst formation and increased joint fluid. Concomitant synovial inflammation can also occur. Degeneration of cartilage is an irreversible process since articular cartilage cannot regenerate; i.e. it cannot reform normal hyaline cartilage.

Trauma is another important cause of articular cartilage damage. Partial thickness cartilage defects generally do not heal. The subchondral plate remains intact with no access to the vascular system so that classic healing and an associated inflammatory response does not tend to occur. Partial thickness chondral injuries tend to remain relatively stable and rarely progress to osteoarthritis. Full thickness chondral injuries are generally associated with a violation of the subchondral plate exposing the injury to the vascular system via the marrow space. This results in initial fibrin clot formation and an inflammatory response with classic healing. The repair tissue, however, consists of fibrocartilage composed predominantly of Type I collagen rather than Type II collagen. This does not have the normal structure and function of hyaline cartilage. This fibrocartilage generally begins to deteriorate within one year resulting in early onset of osteoarthritis.

Overall, hyaline articular cartilage is characterized by a striking inability to heal even the most minor injury. The causes of this inability to regenerate include the relative avascular status of articular cartilage as well as inability of chondrocytes to migrate. Articular cartilage is also noted to be aneural and thus, is not directly the source of pain in the context of articular cartilage damage. Pain in this context is likely caused by forces that act on the subchondral bone, joint capsule, menisci and other supporting structures of the joint.

MR IMAGING OF ARTICULAR CARTILAGE

Early studies prospectively comparing MR imaging with surgical results in the clinical context of internal derangement in the knee demonstrated excellent sensitivity and specificity for MR imaging diagnosis of meniscal tears and ligament disruptions, however, the sensitivity for cartilage damage was demonstrated to be less than 20% (JBJS 1993; 75B: 49-52). In large series internal derangements in the knee are noted to be isolated to articular cartilage in 25-35% of the patients. Clinically, these cartilage abnormalities can mimic the signs and symptoms of meniscal tears. In a series of 31,516 knee arthroscopies, 37% of the patients had isolated chondral injuries without any associated ligament or meniscal pathology (Arthroscopy 1997; 13:456-460). MR imaging reports of the knee in the clinical context of possible internal derangement, therefore, should probably reflect a similar prevalence of isolated articular cartilage damage.

The accuracy of articular cartilage assessment with MR imaging has improved greatly with the recent development of MR imaging sequences designed specifically for hyaline articular cartilage, improvement in dedicated extremity coil design and the advent of so-called high performance MR systems. High performance MR systems generally consist of a high field strength system with high gradient amplitudes (>20 mT/m), high gradient slew rates (>72 mT/m/sec), and high RF receiver bandwidths (>200 kHz). Preliminary data suggests that high field strength MR systems and in particular high performance MR systems are superior to low field MRI for the detection and characterization of articular cartilage abnormalities. For the detection of severe chondromalacia, a series of 155 consecutive knees with arthroscopic surgical correlation was presented at RSNA in 1998. Grade III or IV chondromalacia was present in 22% of the knees. The sensitivity of MR imaging using a 0.5 T low field MRI system was 14% for severe chondromalacia involving the medial femoral condyle and 0% for other sites in the knee. More recently, Bredella et al assessed three-point Dixon chemical shift imaging for evaluating articular cartilage defects in the knee on a low field open magnet (0.35 T). Compared with arthroscopy, the overall sensitivity was 80%, and the specificity was 73%. Most of the errors were in the detection of early chondromalacia. Sixty-five percent of the cartilage abnormalities were graded identically on MR imaging and arthroscopy.

The use of dedicated extremity coils utilizing quadrature and/or phased array technology is important for accurate MR assessment of articular cartilage. A number of so-called cartilage-specific MRI pulse sequences have also been developed which help to assess articular cartilage with varying success. These techniques are listed in Table I.

A laminar pattern of signal intensity alteration may be seen in articular cartilage when high spatial resolution imaging is utilized with a moderate to long echo time due to the variation of T2 across articular cartilage. A very thin lamina of low signal intensity is noted superficially which corresponds with the superficial zone. Subjacent to this zone is a high signal intensity region that corresponds with the transitional zone. The next subjacent low signal intensity lamina corresponds to the radial zone. Recent studies confirm that this laminated appearance of articular cartilage on spin echo and fast spin echo MR images correlates with histologic zones rather than MR artifacts.

The highest accuracies for clinical evaluation of articular cartilage with routinely available pulse sequences have been obtained with 3D T1-weighted spoiled gradient echo imaging with fat suppression and T2-weighted fast spin echo with fat suppression. The fast spin echo techniques are generally preferred by the author due to their relatively shorter imaging times. In a series of patients with chondral injuries in the knee correlated with arthroscopic surgical results, 3D T1-weighted spoiled gradient recalled echo imaging with fat suppression demonstrated a sensitivity of 86%, specificity of 97% and an overall accuracy of 91% for the diagnosis of chondral injuries. In this series, isolated chondral injuries were present in 23% of the patients (AJR 1996; 167: 127-132). In a more recent series, T2-weighted fast spin echo MR imaging with fat saturation was demonstrated to be an accurate and fast technique for detecting and grading articular cartilage defects in the knee (AJR 1999; 172:1073-1080). In this series of 130 consecutive patients who underwent MR imaging of the knee and arthroscopy, there were 86 arthroscopically proven articular cartilage lesions. The sensitivity was 97%, the specificity 98% and the accuracy 98% in the diagnosis of articular cartilage defects. The combination of the axial and coronal planes using this pulse sequence was found to be optimal.

TREATMENT

The reason behind recent interest in MR imaging of articular cartilage is because of the new developments in the treatment of articular cartilage injury. Recent advances in the treatment of articular cartilage injury include chondrocyte transplantation, improved techniques for osteochondral transplantation and development of chondroprotective agents and cartilage growth stimulation factors. A more complete list of treatment strategies is listed in Table II.

It is useful to digress and consider the modulating factors that effect healing of articular cartilage damage. These modulating factors include the size of the chondral defect. As the size of the

Table 1: Cartilage specific MRI pulse sequences

- T2* - wtd GRE with MTC
- DESS
- 3D T1-wtd spoiled GRE with Fat Sat
- T2-wtd FSE with Fat Sat
- Diffusion-wtd imaging (DWI)
- Sodium (Na) imaging
- Driven Equilibrium Fourier Transform (DEFT)
- MR Arthrography
- "Sandwich" Dixon PD SE (low field)

Table 2: Treatment strategies

- Osteotomy
- Debridement
 - Abrasion Chondroplasty
 - Shaving
- Subchondral Plate Penetration
 - Drilling
 - Microfracture Technique
- Tissue Transplantation
 - Osteochondral Allograft
 - Osteochondral Autograft (Mosaicplasty)
 - Perichondrial Grafts
 - Periosteal Grafts
 - Chondrocyte Transplantation
 - Mesenchymal Stem Cell Transplantation
- Synthetic and Biological Matrices/Scaffolds
- Pharmacologic Agents
 - Corticosteroids
 - Hyaluronic Acids
 - Growth Factors
- Laser
- Continuous Passive Motion

chondral defect increases, the healing response decreases. Whether the chondral defect is partial thickness or full thickness also effects healing. Partial thickness defects result in little or no healing as described above. Patient age and weight also effect healing. Once patients become skeletally mature, there is a decreased healing response. Associated injuries, involving the menisci or ligaments in the knee for example, adversely affect healing. Treatment decisions are therefore frequently based on the size of the chondral defect, its location, whether the defect is partial or full thickness, and the presence of associated injuries in the joint. MR imaging can therefore, help to identify patients who may be eligible for one of these new treatment strategies for cartilage damage and may also be useful in the non-invasive follow-up of these treatments.

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