The articular surfaces of synovial joints are covered by hyaline cartilage, which protects the underlying subchondral bones by distributing loads and absorbing shock, maintains low-contact stresses, reduces friction and enables smooth motion of the articulating osseous structures. Damage to the hyaline cartilage usually results from everyday weight-bearing forces or sport activities and might be worsened with additional destabilizing pathology, such as tears (and, thus, subsequent loss of the supportive function) of menisci or labrum.

MR imaging is the method of choice to identify articular cartilage injuries and evaluate the post-operative reconstruction of cartilage repair tissue. Although various experimental techniques, such as T2 mapping, post-contrast T1 mapping, T1 rho imaging, glycosaminoglycan chemical exchange saturation transfer imaging (GAG CEST), and sodium MR imaging, are becoming feasible for the assessment of cartilage tissue architecture, conventional morphologic MR imaging remains the mainstay for the pre- and post-operative evaluation of the articular cartilage (Fig. 1). This chapter describes the structural anatomy of the articular cartilage, along with the respective normal and abnormal imaging appearances and delineates various grading systems and guidelines used in clinical practice for the imaging definitions of cartilage injury (chondrosis). In addition, surgical techniques for cartilage reconstitution are briefly discussed, and instructions are provided for filling in the structured template for the MR imaging description of repaired cartilage.

ANATOMICAL ARCHITECTURE AND PATHOPHYSIOLOGY

The hyaline cartilage is a thin layer of soft tissue, wrapped around complex intra-articular anatomical structures. It is composed of 70% water, 20% collagen, and 5–10% proteoglycans and is very hypocellular since it is only composed of about 4% chondrocytes by wet weight. From superficial (near the joint fluid) to deep (near the subchondral bone), the cartilage is composed of (1) the lamina splendens, which is a thin hypointense protective layer; (2) a superficial layer of horizontal collagen fibers, which provide resistance against shear stress; (3) an intermediate/transitional layer of oblique fibers, which provide resistance against shear and compression stress; (4) a deep layer of radial fibers, which provide resistance against compression stress; and (5) a calcifying/tidemark zone, which tightly bonds the cartilage to the underlying bone.

The cartilage is subject to repetitive forces throughout life, and a normal adult loses 1% to 3% of articular cartilage thickness per year after 30 years of age, a process, which further worsens with the onset of osteoarthritis or secondary insults from acute trauma, infection, or joint instability from...
meniscal ligamentous injuries or inflammatory arthropathies. In general, arthritis-related cartilage defects show irregular and obtuse margins due to repetitive wear and tear whereas acute trauma-related defects are usually focal, isolated and demonstrate well-defined shouldered margins.

Cartilage damage progresses from chondromalacia (softening) to chondrosis (fissures, defects, flaps, delamination, and denudation on one side of the joint) and osteoarthrosis (cartilage defects or loss on both sides of the joint). Cartilage injuries are usually repaired by chondrocytes, which usually form fibrocartilage. The latter, however, is not very resilient in dealing with stresses as compared to the native hyaline cartilage. Full-thickness cartilage loss commonly results in stress changes in the underlying bone, causing pain and decreased range of motion in the affected joint.

On MR imaging, the normal five-layered configuration of hyaline cartilage is rarely visible, except in areas where it is thickest, for example, in patella. It is also only possible if high-resolution techniques combined with joint-specific coils and high-field strength (≥ 3 T) scanners are employed. On conventional (proton density-weighted [PDW] and fat-suppressed PDW [fsPDW]) imaging, the articular cartilage usually features a trilaminar configuration, composed of a low-signal deep layer (tidenark and radial zone), a thicker intermediate to hyperintense middle layer (oblique and horizontal fibers), and a thin low-signal surface layer (lamina splendens). In general, there is a gradual increase in signal from the bone surface to the articular surface. Additionally, the thickness of different layers of the cartilage vary for different surfaces of the articular bony margins (e.g., the radial layer is thicker in the weight-bearing central aspect of the tibia, and the transitional layer is thicker in the peripheral aspect) (Fig. 1).

### IMAGING OF ARTICULAR CARTILAGE PATHOLOGY

Cartilage injury is often a part of osteochondral lesions (OCLs). OCLs refer to one or two lesions on either joint surface, which have resulted from trauma, osteochondritis dissecans (OCD), or insufficiency fracture. More than two to three lesions are generally clubbed together as part of osteoarthrosis. MR imaging is the method of choice for the evaluation of both OCLs and intrasubstance lesions. A number of terms have been used for describing cartilage lesions on MR imaging, and multiple grading schemes have been proposed. Some of these include the Outerbridge Classification (1961), the Osteoarthritis Cartilage Histopathology Grading and Staging (OARSI, 2006), the International Cartilage Repair Society (ICRS) Classification (Table 1), and the Whole Organ MR Imaging Score (WORMS). Remembering and incorporating these ever-changing grading systems is a challenging task for both radiologists and referring physicians. In addition, on MR imaging, a number of lesions of varying severity may be identified, the interobserver variability tends to increase with complicated scoring systems, and a particular scoring system may not be efficient in classifying all such lesions. It is, therefore, better to describe the lesions in each compartment of a joint using accurate terminology rather than trying to fit all lesions into a particular scoring system. The terminology used in common clinical practice for describing cartilage lesions is illustrated in the following paragraphs.

#### Signal Heterogeneity

On MR imaging, signal heterogeneity may be caused by chondromalacia, fibrocartilage formation, cartilage degeneration, or mineralization. In most circumstances, chondromalacia (blistering or softening) is the earliest stage of cartilage injury; although it may be present in the cartilage either focally or diffusely in combination with higher grades of injury. The lesion usually involves the deeper layers of the cartilage and is likely related to fluid imbibition from a not-so-well seen surface fissure. The name derives from soft “malacic” feeling on arthroscopic probing. MR imaging may show focal area(s) of increased fluid-like T2 signal intensity in the deeper layers of the cartilage, with or without associated focal cartilage swelling. It can be differentiated from fissure and defect from the fact that the overlying lamina splendens hypointensity remains preserved. More commonly, the chondromalacic cartilage features diffusely increased signal with loss of the normally observed gradual deep to superficial signal alteration as described above.

Fibrocartilage formation, cartilage degeneration, and/or chondrocalcinosis are the result of subacute/chronic cartilage injury, as the cartilaginous tissue attempts to self-heal by fibrosis or mineralization. MR imaging shows focal or diffuse areas of low signal intensity within the articular cartilage (Figs. 4, 5). It is often difficult to differentiate fibrocartilage from chondrocalcinosis although the latter tends to be more focal and punctate and may additionally show blooming artifact on gradient echo images (Fig. 6). Correlation with plain films may assist in identifying the calcifications, although the lesions are often not visible, due to the low sensitivity and poor soft tissue contrast of conventional radiographs. Large areas of chondrocalcinosis may mimic incomplete discoid meniscus or vacuum in the joint (Fig. 7).

#### Partial-thickness Lesions

Partial-thickness defects qualify under chondrosis, and the term chondromalacia should not be used to describe them. These lesions can show a variety of morphologic appearances such as surface fibrillation, fissures, flaps, defects, or simply thinning. Apart from using these terms, one should describe

<table>
<thead>
<tr>
<th>Grade</th>
<th>Cartilage Key Feature</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>Surface fibrillation and/or superficial laceration</td>
</tr>
<tr>
<td>3</td>
<td>Partial-thickness defect &lt;50% of thickness</td>
</tr>
<tr>
<td>4</td>
<td>Partial-thickness defect &gt;50% of thickness</td>
</tr>
</tbody>
</table>

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**Table 1:** The International Cartilage Repair Society (ICRS) classification

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**Cartilage Repair Society (ICRS) Classification (Table 1), and the Whole Organ MR Imaging Score (WORMS).**
**Fig. 2:** Patellar chondromalacia. Axial images from different cases show diffuse hyperintensity (long arrows) of the lateral patellar facet cartilage (faint in A, prominent in B), which is indicative of chondromalacia. In (B), an additional full-thickness shouldered cartilage defect (short arrow) can be seen at the median patellar ridge.

**Fig. 3:** Trochlear chondromalacia. Axial image reveals a tiny blister-like increased signal (arrow) at the central aspect of the trochlear cartilage.

**Fig. 4:** Multiple patellar cartilage abnormalities. Axial image (A) exhibits linear areas of hyperintensity (short arrows) at the cartilage of both facets, which are indicative of chondromalacia, a hypointense focus (arrowhead) in the medial facet cartilage, which corresponds to fibrocartilage, as well as low-grade cartilage defects (long arrows) at the lateral facet. Axial (B) and coronal (C) reformations from a three-dimensional dual echo steady state sequence render the fibrocartilage focus (arrowheads) and cartilage defect (arrows) more prominent. Chondromalacia is less well seen on these 3D images.

**Fig. 5:** Multifocal patellar chondrosis and signal alterations: Sagittal (A), axial (B), and corresponding sagittal color-coded T2 map (C) show multifocal high-grade patellar cartilage defects (arrows) with background areas of chondromalacia and fibrocartilage formation (arrowheads), not an uncommon finding in patellar malalignment cases.

**Fig. 6:** Chondrocalcinosis. Axial gradient echo image exhibits punctuate foci (arrows) of low signal within the patellar cartilage, which correspond to calcifications.
whether the lesion is less than or more than 50% in thickness, namely low grade or high grade, respectively. If uncertain, one can use the term intermediate grade instead. Surface fibrillation represents an early stage of cartilage loss. On MR images, the cartilage shows minimal surface irregularity (fraying) and loss of the dark layer of the lamina splendens (Fig. 8). Cartilage fissure or flap results from further surface damage. Fissures are identified as tiny linear T2 bright gaps of less than 2 mm in transverse width, which assume a nearly perpendicular orientation to the articular surface (Fig. 9). A flap is formed by an obliquely oriented fissure, which causes elevation of the superficial (<50%) or deep (>50%) semi-separated layers of the articular cartilage (Figs. 10, 11). Cartilage defects are larger gaps with transverse width of greater than 2 mm. Based on the involved thickness, they can also be classified as low- and high grade, similar to fissures and flaps (Figs. 4, 5). In general, acute–subacute trauma related defects are well defined with shouldered margins, show acute angles and are fewer in number and are limited to the trauma sites. The osteoarthritis (OA)-related defects show obtuse margins, are ill-defined and occur at all sites, especially worse in the weight-bearing areas. Cartilage thinning appears as diffuse decrease of the cartilage thickness, with or without focal defects. In an individual joint, comparison with the cartilage thickness of the normal-appearing compartment assists in identifying the abnormality. Sometimes the cartilage may show low-intermediate grade thinning; however, presence of bony changes of sclerosis, cysts, and edema may indicate underlying full-thickness fissuring (Figs. 12–14). Patellofemoral pain syndrome patients will classically show patellofemoral cartilage abnormalities at the opposing surfaces with associated underlying friction-related Hoffa’s fat pad edema (Fig. 15). In addition, these subjects might show excessive or isolated cartilage loss of the far posterior medial or lateral femoral condyles in the tibiofemoral compartments (Fig. 16).

**Full-thickness Lesions**

Full-thickness cartilage fissures or defects are the most severe abnormalities, which rapidly evolve over time and lead to loss of stress shielding of the underlying bone leading to subchondral edema, cysts, and/or sclerosis (Figs. 5, 13, 16–21). The latter manifestations are evident on PDW and fsPDW images: edema is disproportionately apparent on fsPDW images; cysts are proportionately apparent on PDW and fsPDW images as dark/bright lesions on PDW and bright lesions on fsPDW images; sclerosis is proportionately dark.

(text continues on page 138)
Fig. 11: Cartilage injury caused by transient patellar dislocation. Sagittal (A) and axial (B) images exhibit contusion and a high-grade flap (arrows) at the medial patellar facet cartilage with subchondral bony defect.

Fig. 12: Trauma-related shouldered cartilage defect. Coronal images (A, B) reveal a well-defined shouldered defect at the lateral aspect of the medial femoral condyle (arrows).

Fig. 13: Osteoarthritis-related cartilage loss. Coronal (A) and sagittal (B) reformatted images from a three-dimensional sequence show low- to intermediate-grade cartilage defects (short arrows) at the anterior aspect of the talar dome and the opposing surface of the tibia, as a result of chronic injury and resulting OA. Underlying subchondral cysts (long arrows) are presumably caused by thin, nonvisible fissures. Also note the foci of fibrocartilage (arrowheads) formation of the tibial articular cartilage.

Fig. 14: Osteoarthritis-related cartilage lesions. Coronal images (A, B) exhibit areas of high-grade cartilage thinning (arrows) at the medial compartment. Also note medial meniscus degeneration (arrowhead).
Fig. 15: Patellofemoral friction syndrome. High-grade cartilage lesions in a patient with patellofemoral pain. Sagittal image shows superolateral Hoffa's fat pad edema (short arrow), which is indicative of the entity, along with high-grade cartilage thinning (long arrows) at the trochlea and patella, due to chronic friction.

Fig. 16: High-grade cartilage lesions in a patient with patellofemoral pain. Sagittal reformatted three-dimensional images (A, B) demonstrate subchondral cysts (short arrows) at the far posterior aspect of the lateral femoral condyle, which are associated with high-grade thinning of the overlying cartilage (long arrows) and presumably cartilage fissures. This finding is not common in young persons with patellofemoral malalignment and maltracking syndromes.

Fig. 17: Full-thickness cartilage defect. Coronal images (A, B) reveal full-thickness cartilage defect (short arrows) of the lateral talar dome, along with hypointense focus (long arrows) in the adjacent cartilage, signifying degeneration. Findings are similarly appreciated on reconstructions in the oblique (C) and coronal (D) planes from a three-dimensional sequence.
Fig. 18: Osteoarthritis-related patellofemoral cartilage lesions. A: Axial image demonstrates homogeneous high-grade thinning of the lateral facet cartilage, lateral facet trochlea, and full-thickness defect of the median ridge with scattered subchondral cysts (arrows). B: Sagittal image from a different case show a full-thickness cartilage defect (arrow).

Fig. 19: Osteoarthritis-related cartilage lesions. Sagittal images (A, B) reveal high-grade and full-thickness cartilage defects (long arrows) at both articular surfaces of the lateral knee compartment. Also note associated chondral osteophyte formation (short arrows), a high-grade cartilage flap (open arrows) at the far posterior condyle, as well as posterior horn truncation (arrowheads) of the ipsilateral meniscus.

Fig. 20: Osteoarthritis-related cartilage lesions. Sagittal reformations from a three-dimensional sequence reveal broad areas of full-thickness cartilage defects (arrows) at both sides of the medial knee compartment. Also note subchondral sclerosis (arrowhead in A) of the medial plateau and faint edema (arrowhead in B) at the ipsilateral femoral condyle, signifying recurrent bone abutment.

Fig. 21: Trauma-related shouldered cartilage defect. Coronal conventional image (A) and coronal reformatted three-dimensional image (B) demonstrate a shouldered full-thickness cartilage defect (arrows) at the lateral femoral condyle.
on both PDW and fsPDW images. Other reactive bony changes include subchondral insufficiency fracture, articular surface collapse, and chondral or marginal osteophyte formation. Among these lesions exist, cartilage delamination and denudation. Delamination refers to debonding of the articular cartilage from the subchondral bone at the tidemark zone and is regarded as one of the most severe cartilage lesions. MR imaging shows fluid-like signal interposed between the cartilage and the bone, with or without buckling of the delaminated cartilage (Figs. 22, 23). Denudation ranges from broad areas of full-thickness cartilage defects to complete loss of the cartilage from the weight-bearing areas of the bone (Fig. 24). In worst case scenarios, both opposing surfaces of the bone might be completely denuded, leading to bone-on-bone articulation and osteoarthritis.

**Osteochondral Lesions**

The term OCLs refers to one or two lesions of the articular cartilage, which are associated with reactive changes of the underlying subchondral bone. OCLs may be related to meniscal or labral tears a.k.a subchondral insufficiency fractures or stress injuries; acute trauma a.k.a osteochondral fracture; or overactivities of adolescence a.k.a OCD. Osteochondral fractures manifest as one or a combination of findings such as bone marrow edema, partial- or full-thickness我们应该从文章中删除。
**Fig. 25:** Osteochondral fracture caused by crutch injury. Coronal images (A, B) show an osteochondral fracture (arrows) at the superior aspect of the humeral head.

**Fig. 26:** Osteochondral fracture of the tibia. Sagittal images. A: A displaced transverse fracture (arrow) is evident at the anterior aspect of the radial head from recent elbow dislocation. B: Hypointense intra-articular osteochondral bodies (arrows) float within the anterior joint effusion.

**Fig. 27:** Osteochondritis dissecans with in situ body. Anteroposterior (A) and lateral (B) radiographs reveal an osteochondral defect (long arrows) at the medial femoral condyle, with in situ osseous bodies (short arrows). The lesions are better appreciated on the respective coronal (C) and sagittal (D) MR images depicting the unstable lesion with overlying cartilage loss.
**Musculoskeletal MRI Structured Evaluation**

**Fig. 28:** Osteochondritis dissecans of the capitellum. Coronal images (A, B) demonstrate a crescentic subchondral lesion (arrows) in the capitellum, which is surrounded by marrow edema.

**Fig. 29:** Osteochondritis dissecans of the capitellum with in situ body. Coronal image (A) shows a crescentic subchondral lesion (arrow) in the capitellum. On the respective axial images (B, C), there is an osteochondral defect in the capitellum (long arrows), which is filled with an in situ osteochondral body (short arrows). The latter is surrounded by fluid, indicating an unstable lesion.

**Fig. 30:** Osteochondritis dissecans of the knee. Sagittal images (A, B) reveal thickened secondary physis (long arrows) with subchondral cysts at the posterior aspect of the lateral femoral condyle. The overlying cartilage (short arrows) is proud and chondromalacic.
Except in OCLs and OCDs, bone marrow edema and subchondral cysts may appear in various other entities such as trauma (contusions and fractures can evolve into cysts), avulsive changes (in tendon or meniscal root attachments, usually associated with underlying enthesisophyte formation), infection (intraosseous abscesses can mimic cysts), and neoplasms. Features assisting in differentiating cartilage-related cystic changes from the above entities include a bubbly or multiloculated appearance, communication with the articular surface or presence of a neck reaching the articular surface, epicenter located close to the articular surface, irregularity of overlying cartilage or bony endplate, and internal fat, which may be seen in larger cysts, and which reflects seepage of joint fluid around the normal marrow fat. In uncertain cases, contrast administration will reveal enhancement in the periphery of large cysts, mimicking synovial enhancement.

Surgical repair techniques include debridement (smoothening the eroded surface), abrasion arthroplasty (erosion of few millimeters of subchondral bone, causing local bleeding and subsequent development of fibrocartilage-like cover on the defect), and microfracture (multiple perforations to the subchondral bone stimulating the migration of hematopoietic and mesenchymal stem cells to form the new tissue). Microfractures are effective in small injuries and small (<1–2 cm²) cartilage defects, which feature intact subchondral plate (Fig. 31). However, the result is fibrocartilage formation, which is not as strong as the hyaline cartilage in terms of shock-absorbing properties.

Rehabilitation techniques include autologous osteochondral graft osteochondral autograft transfer system (OATS) procedure (graft received from a non–weight-bearing area of the intra-articular bone and transplanted into the cartilage injury defect) (Figs. 32–34), mosaicoplasty (multiple osteochondral autografts bundled like a coaxial cable or cigarette butt to cover a larger area), and synthetic grafts/osteochondral plugs (bioabsorbable devices to press fit into the gap). Regenerative techniques include one-step technique, juvenile (de novo) cartilage implantation (drilling a smooth crater at the OCL site and filling with juvenile cartilage cells covered with glue or periosteal patch on the top) (Figs. 35, 36) and two-step technique, autologous chondrocyte implant (ACL) (harvesting the host articular cartilage from a non–weight-bearing area of the knee, culturing chondrocytes in vitro, and reimplantation at the cartilage defect site, with coverage using periosteal patch or synthetic patch). These techniques can be used for lesions between 1 to 2 and 10 cm². These techniques are being ever evolving and multiple clinical trials are being conducted to test their efficiency. From Asia, there have been case reports of uniform microfracturing of entire femoral condyles or other large areas with stem cell infusion treatments in the joint, that have shown promising early results (Figs. 37, 38).

Postoperative follow-up imaging of the repair cartilage is essential to determine the extent of defect filling, the degree of peripheral integration with the host tissue, the morphologic structure and signal of the repair tissue, and the (text continues on page 145)
Fig. 32: Autologous osteochondral graft procedure. Coronal (A, B) and sagittal (C, D) images show a subchondral fracture (long arrows) of the medial femoral condyle, with minimal surrounding marrow edema and early cyst formation. Also note a horizontal tear (short arrows) of the medial meniscus. The patient underwent autologous osteochondral graft procedure. (Continued in Fig. 33.)

Fig. 33: Postoperative MR examination of the patient in Fig. 32 2 months after the procedure. On coronal (A, B) and sagittal (C, D) images, there is minimal underfilling (>50%) of the osteochondral defect, with the repair tissue (arrows) showing complete coverage of the bone interface, complete cartilage interface in the sagittal plane and incomplete (defect <50%) in the coronal plane, intact surface, and homogeneously normal signal. The subchondral lamina is intact, and there is minimal subchondral edema. Sagittal images (E, F) show the graft harvest site (arrows) at the trochlea. (Continued in Fig. 34.)
**Fig. 34:** Postoperative examination of the patient in Figs. 32 and 33 1 year after the procedure. Compared with the 2-month postoperative images, coronal (A, B) and sagittal (C, D) images now show repaired tissue that is flush with the adjacent cartilage and even less incomplete cartilage interface in the coronal, indicating maturation of the graft (arrows). Also note the postoperative configuration of the medial meniscus (arrowhead). E–G: Sagittal images demonstrate the graft harvest site (arrows), which underwent reconstitution by a combination of hyaline and fibrous cartilage. The formation of cystic changes and increasing edema (depicted in G) suggest deterioration due to stress changes from underlying uncorrected patellar malalignment.

**Fig. 35:** Surgical repair of a talar cartilage lesion with de novo juvenile cartilage cell technique. Coronal images. A: A high-grade cartilage defect (arrow) is evident at the medial aspect of the talar dome with subjacent cysts and edema. B: Postoperative image after de novo cartilage filling of the defect shows mild hypertrophy of the repair tissue, nearly flush with the native cartilage (arrow), which features complete bone and cartilage interfaces, intact surface and internal structure, as well as relatively homogeneous signal. The subchondral lamina is intact. A small area of marrow edema persists.

**Fig. 36:** Surgical repair of a talar cartilage lesion using juvenile cartilage cells. Postoperative sagittal (A) and coronal (B) 3D images show mild hypertrophy (<50%) of the repair tissue, which features incomplete bone and incomplete cartilage interfaces with a flap at the anterior and medial aspect of the lesion on the talar dome (arrows), irregular surface and heterogeneous internal structure, as well as relatively homogeneous signal. The subchondral lamina is intact. There is no marrow edema, joint effusion, or chondral osteophyte formation. Minimal residual subchondral cysts are seen at the posterior aspect of the lesion.
Fig. 37: Multisite drilling large area technique for cartilage repair. Sequential axial (A–F) and sagittal (G–J) images demonstrate multiple uniformly placed microfractures (short arrows) drilled in the subchondral bone at the patella, trochlea, femoral condyles, and lateral tibial plateau for the repair of overlying cartilage lesions (arrowhead). The surgery also included anterior cruciate ligament reconstruction (long arrows). Subsequently, peripheral stem cell harvest and weekly intra-articular injections of stem cells and Hyalgan followed for 6 weeks, along with physiotherapy and continuous passive motion on a daily basis. The results are shown in Fig. 38. (Case courtesy of Dr. Shahrin Merican, MD.)

Fig. 38: Postoperative evaluation of the patient in Fig. 37 9 months after the surgery. Arrows in axial (A, B) and sagittal (C–E) images indicate the drilling sites. In all sites there is mild underfilling of the defects with cartilage, which features complete bone and cartilage interfaces, heterogeneous signal, and mild surface irregularity. The subchondral lamina is intact with small joint effusion and minimal marrow edema in the medial facet of the patella.
integrity of the host cartilage. Recommended at 3 to 6 months, the initial follow-up examination provides assessment of the volume and the integration of the repair tissue. Subsequent imaging in the first postoperative year enables evaluation of the maturation of the graft and identification of any complications. Initially proposed by Marlovits et al., who employed two-dimensional MR sequences, the Magnetic Observation of Cartilage Repair Tissue (MOCART) scoring system is the most widely used technique for monitoring the cartilage repair over serial intervals. The system is based on the analysis of the following nine variables: degree of repair filling, integration of the cartilage repair tissue to the border zone, structure of the surface, structure of the whole repair tissue, signal intensity of the repair tissue, constitution to the subchondral lamina, status of the subchondral bone, possible adhesions, and possible joint effusion. With the increasing clinical utility of high-field scanners and three-dimensional sequences, a modified MOCART score was proposed by Welsch et al. that includes 11 variables (Appendices 1 and 2). In general, bone marrow edema, and cartilage hyperintensity at the repair site should resolve by the first 12 months. Surface irregularity is commonly observed as a normal finding, and the margins of the repair site may normally remain visible for prolonged periods of time. Persistent bone marrow edema beyond 12 to 18 months post-repair, chondral osteophyte or extensive cyst formation, and cartilage delamination are poor prognostic indicators and suggest repair failure. Additional complications include arthrofibrosis and graft hypertrophy. The latter can lead to joint locking and may require debridement.

To conclude, descriptive terminology should be used in cartilage description. One should distinguish trauma-related shouldered defects from OA-related obtuse marginated lesions since the treatments are different. Finally, structured reporting, especially MOCART scoring system, should be used for postoperative cartilage repair tissue.

SUGGESTED READINGS

APPENDIX 1: The Structured Report: Modified MOCART

SITE: [<Femoral condyle> <Tibial plateau> <Patella>]

1. DEFECT FILL (degree of defect repair and filling of the defect in relation to the adjacent cartilage):
   - <Flush with the adjacent cartilage>
   - <Hypertrophy (proud as compared to the adjacent cartilage)>
   - <Underfilling (below the level of the adjacent cartilage)>
   Additional qualifier: [<Thickness less than 50% of the adjacent cartilage>]
   - <Thickness more than 50% of the adjacent cartilage>]

2. CARTILAGE INTERFACE (integration with adjacent cartilage to border zone in two planes)
   - Sagittal (femur, patella, trochlea, tibia):
     - <Demarcating split-like border visible>
     - <Complete>
     - <Defect less than 50%>
     - <Defect more than 50%>
   - Additional qualifier: Whole area of cartilage repair: [<Integration less than 50%>]
     - <Integration more than 50%>]
   - Coronal (femur, tibia); axial (patella, trochlea):
     - <Demarcating split-like border visible>
     - <Complete>
     - <Defect less than 50%>
     - <Defect more than 50%>
   - Additional qualifier: Whole area of cartilage repair: [<Integration less than 50%>]
     - <Integration more than 50%>]

3. BONE INTERFACE (integration of the transplant to the subchondral bone; integration of a possible periosteal flap):
   - <Complete>
   - <Partial delamination>
   - <Complete delamination>
   Additional qualifier: Whole area of cartilage repair: [<Delamination less than 50%>]
   - <Delamination more than 50%>]

4. SURFACE (constitution of the surface of the repair tissue):
   - <Intact>
   - <Less than 50% depth damage>
   - <More than 50% depth damage>
   - <Adhesions>
   Additional qualifier: Whole area of cartilage repair: [<Less than 50%>]
   - <More than 50%>]

5. STRUCTURE (constitution of the repair tissue):
   - <Homogeneous>
   - <Inhomogeneous / Cleft formation>
   Additional qualifier: Whole area of cartilage repair: [<Less than 50%>]
   - <More than 50%>]

6. SIGNAL INTENSITY (intensity of MR signal of the repair tissue in comparison to the adjacent cartilage:
   - <Normal>
   - <Nearly normal>
   - <Abnormal>
   Additional qualifier: Whole area of cartilage repair: [<Less than 50%>]
   - <More than 50%>]

7. SUBCHONDRAL LAMINA (constitution of the subchondral lamina):
   - <Intact>
   - <Not intact>
   Additional qualifier: Whole area of cartilage repair: [<Less than 50%>]
   - <More than 50%>]

8. CHONDRAL OSTEOPHYES (osteoaphyes within the cartilage repair area):
   - <Absent>
   - <Osteophytes less than 50% of repair tissue>
   - <Osteophytes more than 50% of repair tissue>

9. BONE MARROW EDEMA (maximum size and localization in relation to the cartilage repair tissue and other alterations assessed in the 3D MOCART score: small = less than 1 cm, medium = less than 2 cm, large = less than 4 cm):
   - <Absent>
   - <Small>
   - <Medium>
   - <Large>
   - <Diffuse>

10. SUBCHONDRAL BONE (constitution of the subchondral bone):
    - <Intact>
    - <Granulation tissue>
    - <Cyst formation>
    Additional qualifier: Whole area of cartilage repair: [<Less than 50%>]
    - <More than 50%>]

11. EFFUSION (approximate size of joint effusion visualized in all planes):
    - <Absent>
    - <Small>
    - <Medium>
    - <Large>

IMPRESSION:

1. <>
APPENDIX 2: Sample Completed Structured Report: Modified MOCART: ABNORMAL/POSITIVE

SITE: Lateral femoral condyle—central weight-bearing portion

1. DEFECT FILL (degree of defect repair and filling of the defect in relation to the adjacent cartilage):
   - Underfilling
   Additional qualifier: Thickness more than 50% of the adjacent cartilage

2. CARTILAGE INTERFACE (integration with adjacent cartilage to border zone in two planes)
   - Sagittal: Demarcating split-like border visible and the defect is less than 50%
   - Coronal: Complete
   Additional qualifier: Whole area of cartilage repair: Integration more than 50%

3. BONE INTERFACE (integration of the transplant to the subchondral bone; integration of a possible periosteal flap): Complete

4. SURFACE (constitution of the surface of the repair tissue): Intact

5. STRUCTURE (constitution of the repair tissue): Homogeneous

6. SIGNAL INTENSITY (intensity of MR signal of the repair tissue in comparison to the adjacent cartilage: Nearly normal = slight areas of signal alterations)

7. SUBCHONDAL LAMINA (constitution of the subchondral lamina): Intact

8. CHONDRAL OSTEOPHYTES (osteophytes within the cartilage repair area): Absent

9. BONE MARROW EDEMA (maximum size and localization in relation to the cartilage repair tissue and other alterations assessed in the 3D MOCART score: small = less than 1 cm, medium = less than 2 cm, large = less than 4 cm): Small

10. SUBCHONDRAL BONE (constitution of the subchondral bone): Intact

11. EFFUSION (approximate size of joint effusion visualized in all planes): Small

IMPRESSION:

1. Status post OATS procedure lateral femoral condyle, satisfactory result with mild underfilling. No subchondral exposure or delamination.
2. Small joint effusion.