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Narrative Review

Cartilage injury patterns in the professional athlete

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A R T I C L E I N F O

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A B S T R A C T

In this article, the authors review various types of cartilage injury patterns observed on magnetic resonance imaging (MRI) in professional athletes. Several examples of commonly injured joints are presented to showcase the importance of MRI in both the initial detection and characterization of cartilage lesions and the ensuing postoperative success of repair and/or restoration procedures in this unique patient population. Obtaining high-quality MRI studies, thoroughly understanding articular cartilage anatomy and pathology, and familiarizing oneself with the typical mechanisms of injury in various sports are of paramount importance to both radiologists and sports medicine clinicians alike. The authors aim to provide the reader with the foundational tools for approaching articular cartilage injuries in elite athletes and adding value to the diagnosis and treatment of these patients.

Introduction

Articular cartilage injuries are common in elite athletes, particularly given the hypercompetitive nature of modern professional sports.[1–4] Professional athletes are particularly susceptible to cartilage injuries due to repetitive movements in the setting of mechanical overload or sudden explosive movements (such as pivoting, jumping, twisting, or stopping), or secondary to an acute traumatic contact injury during play. Diagnostic imaging, particularly since the advent of high-field-strength magnetic resonance imaging (MRI), plays a critical role in the detection, classification, and grading of articular cartilage injuries, and, in combination with clinical and physical examination findings, helps to determine the most effective treatment plan. Timely identification and treatment of articular cartilage injuries is paramount, as this directly affects the athlete's return to play (ideally at the same level as prior to injury) and greatly impacts their career longevity, potential financial compensation, and, most importantly, long-term health and functionality.[5–7]

MRI is the best noninvasive imaging test to evaluate the morphology and composition of articular cartilage. Proton density (PD) and T2-weighted fast spin echo sequences, as well as T1-weighted spoiled gradient echo sequences, are best suited to evaluate the articular cartilage. Fat saturation is utilized with a fast spin echo to decrease chemical shift artifacts and improve the image contrast between articular cartilage and subchondral bone. Other advanced MRI techniques, such as T1, T2, and T2* mapping, are used to evaluate the internal composition of the cartilage matrix, offering a method to detect biochemical changes in articular cartilage earlier than routine MRI sequences.[8,9] Such advanced techniques are typically not necessary in the evaluation of the professional athlete, particularly in the acute setting, as routine MRI sequences are almost always sufficient to make a diagnosis. An additional critical parameter for image acquisition is magnet field strength. Magnet field strengths of both 1.5 Tesla (1.5 T) and 3.0 Tesla (3 T)

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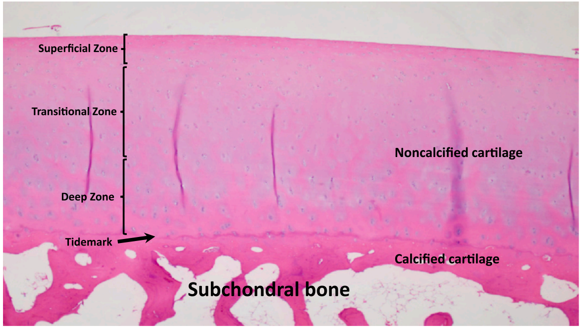

Fig. 1. A histological slide showing normal articular cartilage separated into noncalcified and calcified zones by the tidemark. The tidemark is important due to it being the site of chondral delamination lesions. The calcified cartilage is attached to the underlying cortical bone, which together form the subchondral bone plate. Image courtesy of Dr Wei Jiang, Philadelphia, PA.

are considered adequate for the evaluation of cartilage abnormalities, though 3 T systems offer increased signal-to-noise ratio with better spatial and temporal resolution, as well as decreased scan time.[10] However, there are potential drawbacks to higher field strength imaging at 3 T, including increases in chemical shift artifact and susceptibility artifact in the postoperative setting. For most clinical applications, newer 1.5 T systems with radiofrequency coils optimized for orthopedic imaging provide an excellent assessment of articular cartilage.

At institutions working with professional sports teams, the radiologist plays the important role of an “athlete imaging manager” to ensure that the best workflow is in place for the imaging and interpretation of studies performed on elite athletes. Professional athletes must be imaged discretely and expeditiously with real-time quality assurance to ensure the study is diagnostic and the injury is fully imaged. Imaging findings must then be securely communicated to the team physician, who, together with the other medical specialists and coaching staff, will ultimately determine the treatment plan and return to play.

Types of cartilage injuries on MRI

When treating professional athletes, it is requisite to understand the normal articular cartilage anatomy and the proper nomenclature for describing chondral lesions. At the microscopic level, articular cartilage is made up of collagen, proteoglycans, water molecules, and chondrocytes bound together by hydrogen bonds. At a macroscopic level, articular cartilage can be separated into “uncalcified cartilage” and “calcified cartilage,” with the interface between these 2 regions referred to as the “tidemark.” The calcified cartilage is attached to the underlying subchondral bone via the subchondral bone plate. The uncalcified cartilage is further separated into different zones based on the orientation of the collagen fibers: the superficial zone (fibers oriented parallel to the cartilage surface; very thin), the transitional zone (fibers oriented randomly relative to the cartilage surface; moderate proteoglycan concentration), and the radial zone (fibers oriented perpendicularly relative to the subchondral bone; high proteoglycan concentration) (Fig. 1). It is important to note that articular cartilage has no intrinsic vascular or lymphatic supply and thus must rely on diffusion of nutrients from the synovial fluid and the extracellular space of the subchondral bone.

On MRI, articular cartilage lesions are described in terms of their size, location, and depth. Correctly describing these features is paramount in the case of professional athletes, as this ensures clear communication with other medical professionals and will ultimately help guide the treatment plan. Lesion depth may be categorized as low-grade partial thickness ($< 50\%$), high-grade partial thickness ($\geq 50\%$), or full thickness with exposed subchondral bone. The clinical grading of articular cartilage injuries on MRI is based on modified versions of the classification system originally described by Outerbridge at arthroscopy, the gold standard for evaluation of articular cartilage. The most commonly used grading system, and the one adopted by the International Cartilage Regeneration and Joint Preservation Society (ICRS), categorizes cartilage into grades 0 to IV.[11] Additional secondary findings of chondrosis in the subchondral bone, namely bone marrow edema and cystic change, should be noted. Other concurrent internal derangement lesions, such as meniscal tears and ligamentous injuries, are critically important to recognize in the professional athlete, as these inherently destabilize the knee.

Low-grade chondrosis is a broad descriptive term, which includes cartilage heterogeneity and edema, cartilage thinning, and cartilage fraying—abnormalities that are commonly encountered in professional athletes (Fig. 2A and B). Cartilage heterogeneity and edema likely correspond to grade I ICRS lesions (ie, cartilage softening and indentation on arthroscopic probing), although it is important to note that there may be inherent heterogeneous signal in normal articular cartilage due to the stratified microarchitecture and variable proteoglycan content. Chondral “fibrillation” refers to the irregularity of the articular cartilage surface with associated fraying, splitting, and/or erosion (Fig. 3A and B). These lesions are superficial in nature and affect less than 50% of the cartilage depth (ICRS grade I or II). A chondral “fissure” describes a break in the cartilage surface, which can be of variable depth (Fig. 4A and B). The depth of the fissure (ie, low-grade or high-grade partial thickness or full thickness) determines the ICRS grade (II, III, or IV). Although these aforementioned lesions may be asymptomatic in the professional athlete, the findings are inherently pathologic and reflective of cartilage injury and may progress to larger or higher-grade cartilage lesions in the future.[12,13] Once a

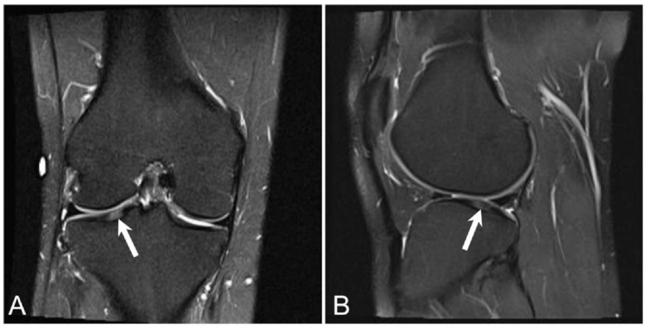

Fig. 2. Elite football defensive end presents for a baseline knee MRI as part of a pre-employment physical. Coronal T2 FS (**A**) and sagittal T2 FS (**B**) MR images show an area of cartilage edema and heterogeneity (arrow) at the medial aspect of the lateral tibial plateau. These low-grade chondral lesions may be reflective of chondropenia and occur in the professional athlete over time due to the intense, repetitive forces experienced at the weightbearing joint surface during play. Although such lesions may be incidental and asymptomatic, they can progress to higher-grade chondral lesions in the future. Anecdotally, this finding at the lateral tibial plateau is frequently seen and may be due to normal variations in the hyaline cartilage composition. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging.

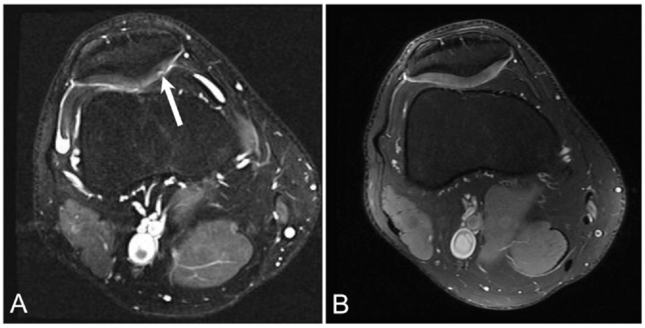

Fig. 3. Elite basketball center imaged following an injury during a game. Axial T2 FS MR image (**A**) of the knee demonstrates focal low-grade cartilage fissuring/fraying at the medial patellar facet (arrow). This was an incidental finding unrelated to the acute injury but new compared to the axial PD FS MR image (**B**) from an MRI obtained 2 years prior. This case also highlights the difference in the appearance of articular cartilage on T2 and PD sequences with fat suppression. Note that the internal cartilage composition is better evaluated on the PD sequence due to the higher spatial resolution and intermediate weighting. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging; PD, proton density.

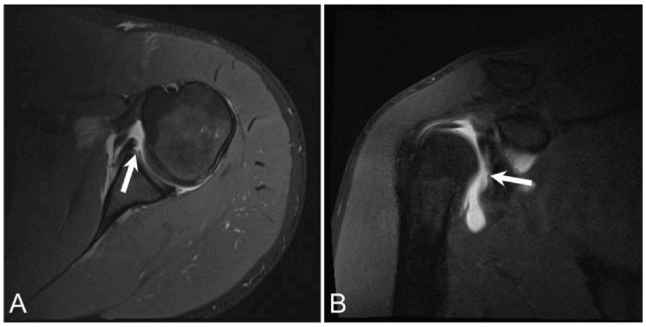

Fig. 4. Professional hockey player with a shoulder injury sustained during a game 2 days prior. Axial PD FS (**A**) and coronal T1 FS (**B**) MR arthrogram images of the shoulder demonstrate an area of partial-thickness chondral fissuring at the anteroinferior glenoid (arrow in **A** and **B**). No shoulder dislocation was noted in this case. FS, fat saturation; MR, magnetic resonance; PD, proton density.

chondral lesion is large enough to be measured in 2 dimensions on MRI, it is no longer considered a cartilage fissure and is better described as a "focal cartilage defect" (Fig. 5A and B).

Rotational and shearing forces with certain movements over a localized area of cartilage can result in a "delamination" injury, which is defined by the separation of the cartilage from the underlying subchondral bone, thought to be at the level of the tidemark.[14] Cartilage delamination injuries can be devastating in the professional athlete (Fig. 6A-C). Chondral "blistering" is a type of delamination injury with focal bulging of the superficial cartilage surface, usually in contiguity with a deeper underlying cartilage lesion

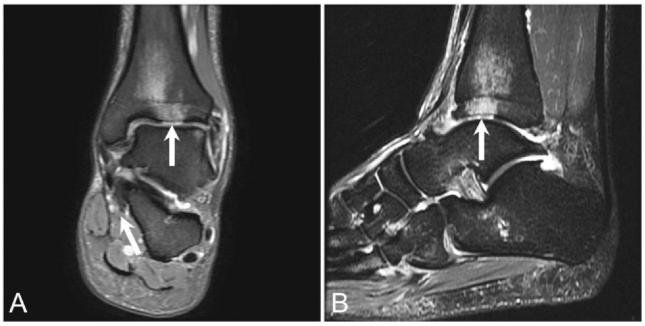

Fig. 5. Professional basketball power forward presents for a baseline MRI before the start of the season. Coronal T2 FS (**A**) and sagittal short tau inversion recovery (STIR) (**B**) MR images of the ankle show a large measurable full-thickness cartilage defect (arrow in **A** and **B**) at the anterior tibial plafond with surrounding subchondral bone marrow edema extending into the distal tibia. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging.

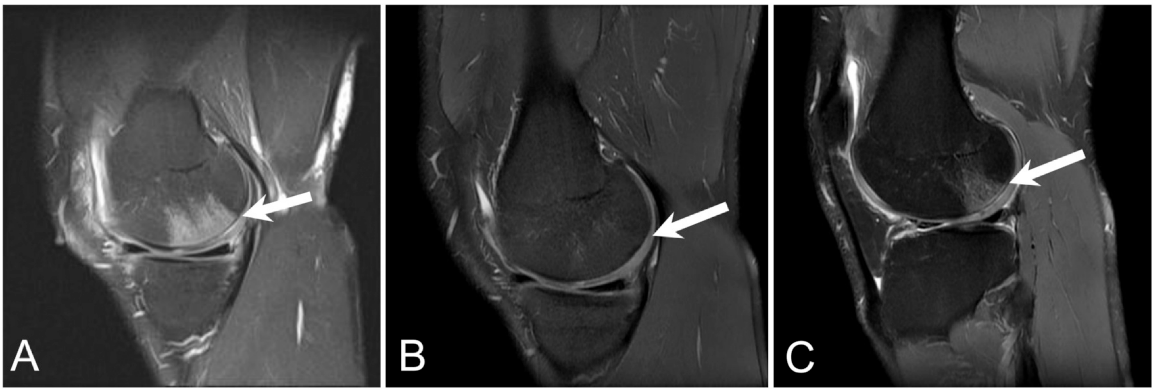

Fig. 6. Elite basketball player presents with medial knee pain. Sagittal T2 FS MR image (**A**) shows extensive subchondral bone marrow edema at the medial femoral condyle secondary to a large overlying, unstable delaminating cartilage lesion (arrow). Sagittal T2 FS MR image (**B**) from a repeat knee MRI obtained 2 months later after conservative treatment shows significant interval improvement (arrow), with the region of delamination no longer visualized and complete resolution of the subchondral bone marrow edema. Unfortunately, the sagittal T2 FS MR image (**C**) from a follow-up knee MRI performed a few months later shows a new delaminating cartilage lesion at the weightbearing *lateral* femoral condyle (arrow) with accompanying subchondral bone marrow edema. Although rare and infrequent in the general population, the altered biomechanics from stress overload at the weightbearing joint surfaces may predispose athletes to an increased risk of delaminating chondral injuries. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging.

(Fig. 7). Delamination type injuries are classified as ICRS grade III and may be occult at arthroscopy if the overlying superficial cartilage is intact.[15] These lesions are important not to miss because they may eventually progress to full-thickness defects. Shear injuries with delamination can also result in displaced intra-articular cartilage fragments or osteochondral fragments (cartilage still attached to the subchondral bone), depending on the degree and exact mechanism of the injury (Fig. 8A and B). For example, this type of injury commonly occurs during transient lateral patellar dislocation due to the unnatural shear forces generated. A chondral "flap" is a tear in the articular cartilage that extends from a delaminating lesion to the cartilage surface, remaining only partially attached (Fig. 9A and B). A cartilage flap may displace or propagate with movement, thus representing a potentially unstable ICRS grade III lesion.[14]

Osteochondral lesions (OCLs) are a group of injuries involving both the cartilage and the underlying subchondral bone. These lesions are seen commonly in various joints in the professional athlete, including the elbow, knee, and ankle (Fig. 10A and B). A staging system for MRI findings is utilized to grade the osteochondral injury and stability and help determine the appropriate treatment. The term "osteochondritis dissecans" (OCD) is generally used to describe the juvenile form of an OCL of a similar pathoetiology and is, therefore, primarily diagnosed in children and adolescents. OCD lesions may heal spontaneously without long-term sequelae or may result in osteochondral fragmentation and/or displacement (Fig. 11A and B). The ICRS has also put forth a classification system to describe OCD lesions.[11] Both OCL and OCD lesions may be either acute or subacute/chronic in etiology, depending on the exact mechanism of injury. Likewise, these lesions may result in long-term pathological changes in the subchondral bone. It is important to acknowledge that OCL lesions may be difficult (or impossible) to distinguish from other ICRS grade IV cartilage lesions, with the former term typically invoked when the lesion is solitary, focal, and measurable, particularly in younger athletes. However, there is significant overlap in the imaging features and management of both lesions in the professional athlete.

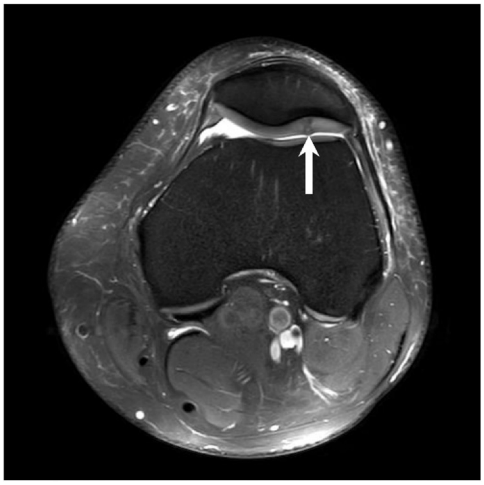

Fig. 7. Elite baseball player presents with 2 weeks of anterior knee pain. Axial PD FS MR image of the knee demonstrates a chondral blister lesion with focal bulging of the superficial cartilage surface at the lateral patellar facet. Though such lesions may appear to be superficial, they extend to the level of the tidemark and can progress to unstable cartilage lesions over time. FS, fat saturation; MR, magnetic resonance; PD, proton density.

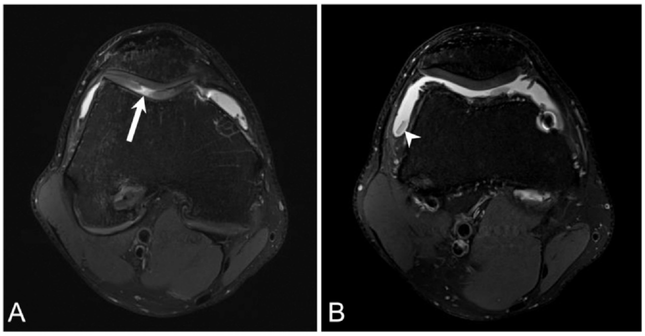

Fig. 8. Professional hockey defenseman presents with lateral knee pain. Axial PD FS MR image (**A**) demonstrates a large full-thickness cartilage defect with adjacent delamination at the lateral aspect of the femoral trochlea (arrow). There is a corresponding intra-articular chondral body in the lateral joint recess (arrowhead) seen a few slices caudal to the level of the chondral defect (**B**). The susceptibility artifact noted along the medial femoral trochlea is related to the patient's prior ACL reconstruction surgery. ACL, anterior cruciate ligament; FS, fat saturation; MR, magnetic resonance; PD, proton density.

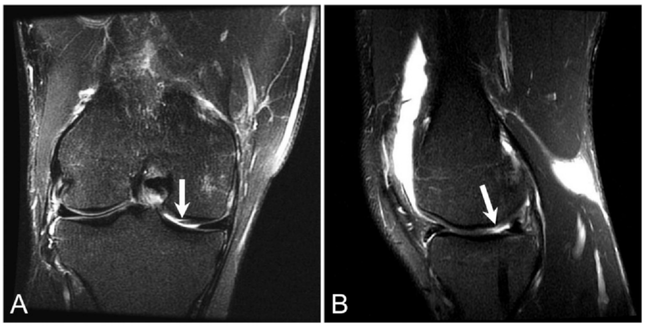

Fig. 9. Elite baseball player presents with right posteromedial knee soreness. Coronal T2 FS (**A**) and sagittal T2 FS (**B**) MR images show a delaminating high-grade partial-thickness chondral flap at the central weightbearing aspect of the medial femoral condyle, an unstable lesion. FS, fat saturation; MR, magnetic resonance.

The end stage of all articular cartilage damage over time is osteoarthritis, which occurs more rapidly in professional athletes due to intense and repetitive forces beyond normal physiologic limits.[16–19] The imaging findings of osteoarthritis are well known and ultimately signify no further utility in attempting repair or restoration of the native joint cartilage (Fig. 12A and B).

Fig. 10. Competitive athlete presents with acute-on-chronic right elbow pain. Sagittal T2 FS (**A**) and coronal T2 FS (**B**) MR images show a large unstable osteochondral lesion, with an osteochondral defect at the capitellum (arrow in **A** and **B**) and the corresponding displaced osteochondral fragment in the anterior joint recess (arrowhead in **A**). There is an accompanying large elbow joint effusion. FS, fat saturation; MR, magnetic resonance.

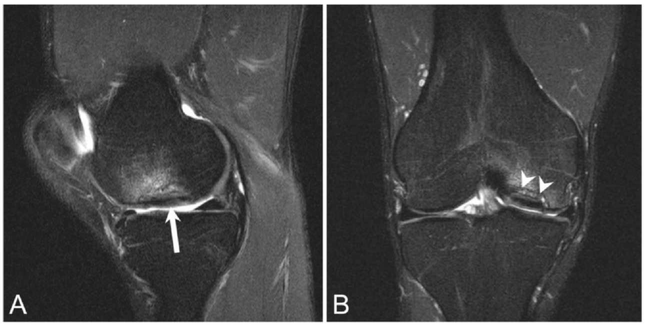

Fig. 11. Elite hockey player presents with right knee pain for 1 day after suffering an in-game injury. Sagittal T2 FS (**A**) and coronal T2 FS (**B**) MR images demonstrate a large OCL at the weightbearing medial femoral condyle (arrow) with associated subchondral bone marrow edema and several small cysts undermining the lesion (arrowheads), suggesting instability. The cartilage overlying the OCL demonstrates heterogeneity and mild incongruity with the adjacent normal articular cartilage. FS, fat saturation; MR, magnetic resonance; OCL, osteochondral lesion.

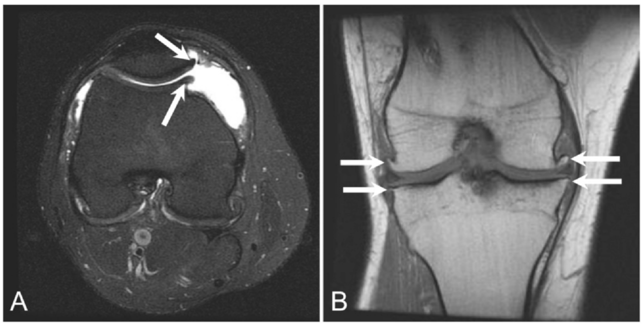

Fig. 12. Professional football defensive lineman without acute injury presents for evaluation of right knee pain. Axial T2 FS (**A**) and coronal T1 (**B**) MR images demonstrate tricompartmental knee joint osteoarthritis with associated marginal osteophyte formation (arrows in **A** and **B**). FS, fat saturation; MR, magnetic resonance.

Common cartilage injury patterns in professional athletes

Cartilage injury patterns in the professional athlete depend on a myriad of factors, including the mechanism of injury and/or overuse, type of sport, and the individual player's position and physique. All joints are inherently different in terms of weightbearing

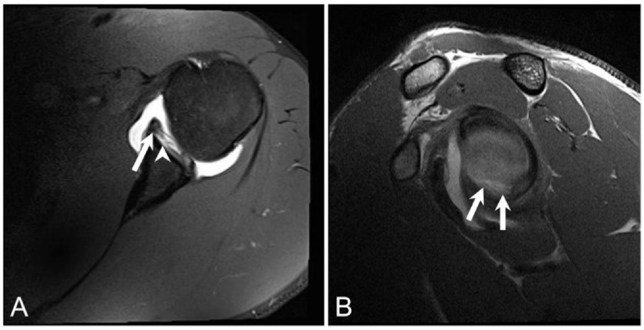

Fig. 13. Professional basketball shooting guard presents with left shoulder pain following a subluxation event 1 day prior. Axial T1 FS MR arthrogram image (**A**) shows a labral tear at the anteroinferior glenoid (arrow) and an associated high-grade partial-thickness cartilage defect with a delaminating component (arrowhead). The sagittal T1 FS MR arthrogram image (**B**) demonstrates the full extent of the lesion (arrows). These findings are consistent with a GLAD lesion. FS, fat saturation; GLAD, glenolabral articular disruption; MR, magnetic resonance.

status, degree of normal motion, stability, and surrounding ligamentous and tendinous support structures. More importantly, the thickness of the articular cartilage in various joints has significant variability. For example, the knee joint is a weightbearing joint with ample cartilage stock measuring 3 to 4 mm.[20] In nonweightbearing joints, isolated cartilage injuries may be more difficult to identify on MRI, such as in the shoulder and wrist, where there is a less robust cartilage stock.[21,22] It is often the case that cartilage injuries in the elite athlete are seen in association with an accompanying ligamentous or osseous injury, though they may also occur in isolation.

Articular cartilage injuries at the shoulder in the competitive athlete can be seen in sports involving contact and collision, such as football and rugby, usually after the athlete experiences a traumatic twisting injury or a subluxation/dislocation.[23] The glenolabral articular disruption lesion usually occurs in the setting of anterior shoulder dislocation or trauma with the shoulder in abduction and external rotation, in which there is tearing of the anteroinferior labrum with associated disruption of the adjacent glenoid articular cartilage (Fig. 13A and B).[24] Although initially thought to be stable, these lesions are now considered unstable and predispose to future recurrent shoulder dislocations.[25,26] Posterior glenoid labral tears, whether acute or chronic/degenerative, are also commonly seen in athletes and may result in accompanying glenohumeral joint chondrosis, preferentially at the posterior glenoid articular cartilage (Fig. 14).[27,28] The predisposition to cartilage injury with posterior labral tears is felt to be secondary to focal intense shear forces at the posterior glenoid/labrum encountered during certain athletic activities, for example, in football linemen when the athlete's shoulder is in 90° of flexion and the elbows are locked in anticipation of the incoming contact.[29]

Chondral injuries at the elbow are mostly seen in athletes who experience repetitive stress at the elbow joint, such as baseball pitchers, other overhead throwing athletes, tennis and volleyball players, and gymnasts.[30] OCD is a well-known lesion at the lateral elbow, usually at the capitellum, which is thought to be secondary to deficient vascular supply in the setting of repetitive overhead or upper extremity weightbearing activities in the adolescent athlete.[31,32] Although OCD lesions are usually self-limited with conservative treatment, early identification is important to avoid lesion progression and instability. OCD lesions may become unstable due to disruption of the osteochondral surface, ultimately leading to osseous fragmentation and development of intra-articular bodies

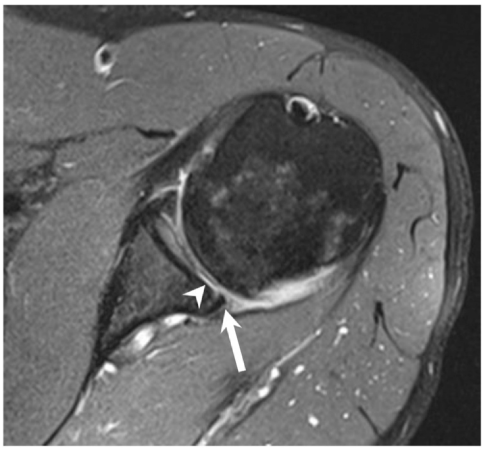

Fig. 14. Elite baseball pitcher presents with shoulder pain at the conclusion of the baseball season. Axial PD FS MR image demonstrates tearing of the posterior labrum (arrow), which destabilizes the glenohumeral joint. There is associated high-grade cartilage loss along the posterior glenoid (arrowhead). FS, fat saturation; MR, magnetic resonance; PD, proton density.

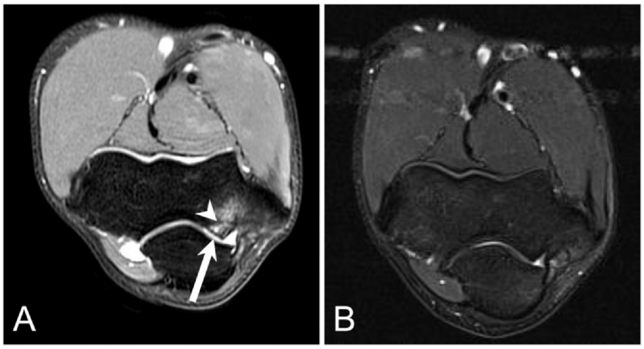

Fig. 15. Professional baseball pitcher presents with medial-sided elbow pain and burning with throwing. Axial PD FS MR arthrogram image (**A**) demonstrates high-grade chondrosis involving the posteromedial trochlea (arrow) with underlying subchondral bone marrow edema and cystic change (arrowhead), findings typical for posteromedial impingement. These findings developed over the duration of this athlete's pitching career, as a T2 axial FS MR image (**B**) from baseline elbow MRI obtained 3 years prior was normal. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging; PD, proton density.

(see Fig. 11). In the adult athlete, OCLs may develop due to repetitive compression and shear forces at the capitellar articular surface, which, as compared to OCD lesions, are more likely to be unstable. Imaging plays a critical role in identifying features that suggest instability, including prominent subchondral cystic change, fluid interposed at the parent-progeny interface, sclerosis along the rim of the lesion, or displacement of an osteochondral fragment. Posteromedial impingement, also referred to as valgus extension overload syndrome or "pitcher's elbow," is another elbow condition due to valgus stress and is typically seen in baseball pitchers.[33,34] Posteromedial impingement occurs secondary to repetitive overuse in the background of an insufficient ulnar collateral ligament, which results in transmission of increased shear forces to the ulnotrochlear cartilage posteromedially, with ensuing osteochondral abnormalities and synovitis (Fig. 15A and B).[33]

The cartilage at the hip is of special interest in the professional athlete due to its intimate relationship with femoroacetabular impingement (FAI).[34–36] Indeed, the majority of chondral lesions seen within the hip are at the anterosuperior acetabulum adjacent to the site of acetabular labral tears in the setting of FAI.[36,37] In FAI, morphologic osseous abnormalities, such as acetabular overcoverage (pincer) or asphericity of the femoral head (cam), result in abnormal contact and damage to the labrum and articular cartilage during certain normal joint motions.[34] FAI is particularly accentuated with movements that place the hip in flexion and internal rotation, as seen with the defensive positioning of hockey goalies.[38,39] Given the prevalence of cam-type FAI morphology in hockey players regardless of position, it has also been posited that skating itself may result in bony overgrowth and the development of cam lesions in these athletes.[40]

In many ways, the knee joint is the paradigm for defining the imaging features of articular cartilage injuries, given the inherent thickness of the cartilage and it being the most frequently injured joint in many different sports, including football, basketball, and soccer.[3,4,41] In addition to the numerous previous examples of knee joint chondrosis in professional athletes due to various mechanisms of injury, transient patellar dislocations may result in acute cartilage shear injuries. Transient patellar dislocations usually occur during an acute traumatic contact injury with the knee in a fixed position. As the patella subluxes laterally along the anterior margin of the lateral femoral condyle, a characteristic osseous contusion pattern occurs, and associated shearing forces on the

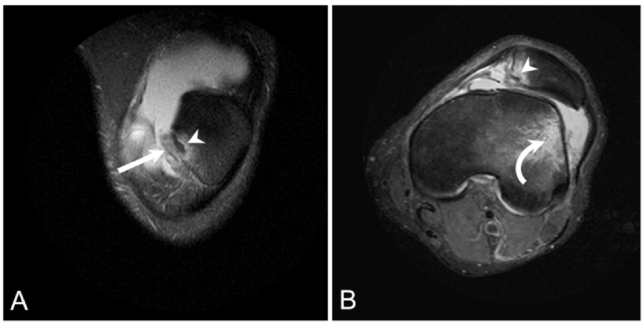

Fig. 16. Competitive soccer player presents for an MRI following a knee contact injury. Coronal T2 FS (**A**) and axial T2 FS (**B**) MR images through the patellofemoral compartment show an osteochondral impaction fracture at the inferomedial patella (arrowhead in **A** and **B**) with an adjacent small, mildly displaced osteochondral fragment (arrow in **A**). There is an associated acute osseous contusion (ie, "bone bruise") along the anterior lateral femoral condyle (curved arrow in **B**), confirming the diagnosis of a transient lateral patellar dislocation. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging.

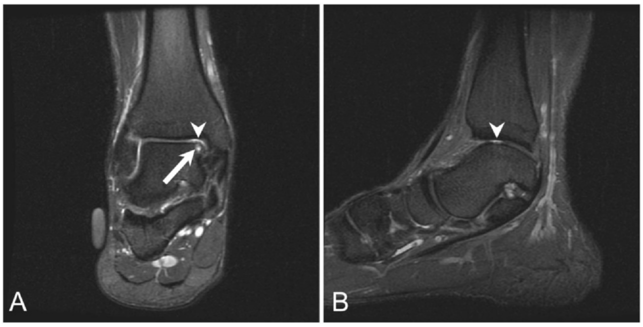

Fig. 17. Elite football wide receiver with a history of recurrent ankle sprains in the past. Coronal T2 FS (**A**) and sagittal STIR (**B**) MR images show a small, stable OCL at the medial talar dome with accompanying mild subchondral bone marrow changes (arrow in **A**) and a reciprocal focal full-thickness cartilage defect at the medial tibial plafond (arrowhead in **A** and **B**). FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging; OCL, osteochondral lesion; STIR, short tau inversion recovery.

articular cartilage may result in displaced cartilage or osteochondral fragment (Fig. 16A and B). Other commonly encountered cartilage injuries in the knee include cartilage fissuring, delamination, flap formation, and OCLs.

Ankle joint cartilage injuries typically result from microtrauma to the articular surface in the background of joint instability, secondary to prior ligamentous and/or osseous trauma, especially in sports with direct contact (eg, tackling in soccer).[41–45] Over time, joint instability and recurrent trauma result in progressive cartilage damage. This may ultimately result in the development of an OCL (Fig. 17A and B). OCLs in the ankle are often found at the medial or lateral aspect of the talar dome due to shearing forces generated during ankle inversion, axial loading, and dorsiflexion (lateral) or plantarflexion (medial).[46,47] Hindfoot alignment may also play a role. Early surgical intervention in OCLs may be necessary to avoid progression to lesion instability and eventual intra-articular body formation.

Postoperative imaging of cartilage repair

Once a chondral lesion is diagnosed on imaging in the professional athlete, the next step is to determine the treatment and, more specifically, to decide whether management will be surgical or nonsurgical. Beyond the return-to-play timeline for the athlete, the size of the cartilage lesion on imaging (either $< 2\,cm^2$ or $> 2\,cm^2$) dictates the available surgical treatment options. These options include chondroplasty and debridement, which are usually reserved for small ($< 2\,cm^2$) partial-thickness defects (Fig. 18A and B). Microfracture or drilling procedures stimulate the marrow at the site of the small ($< 2\,cm^2$) chondral defect, thus promoting the filling of the defect with fibrocartilage (Fig. 19A-C). Although fibrocartilage is inferior to hyaline cartilage, it is often sufficient for the treatment of small lesions.[48,49] Other options include osteochondral autologous transplantation and mosaicplasty, where osteochondral plugs from a nonweightbearing surface of the joint are placed into small or medium-sized chondral defects. For larger lesions ($> 2\,cm^2$), osteochondral allograft is preferred, and osteochondral plugs are instead harvested from cadaveric donor tissue. Autologous chondrocyte implantation and matrix-associated autologous chondrocyte implantation are 2-step techniques whereby the athlete's cartilage is harvested and chondrocytes are isolated ex vivo and later implanted into the cartilage defect. Beyond these known reparative/restorative procedures, which are available to both athletes and nonathletes alike, experimental techniques such as platelet-rich plasma or stem cell injections may be offered to professional athletes in the appropriate clinical setting (Fig. 20A and B).

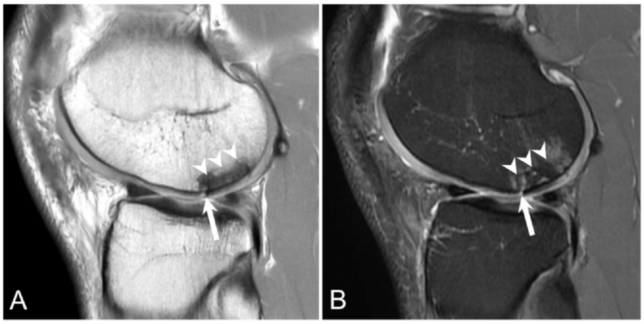

Fig. 18. Professional football linebacker presents for pre-employment physical. Sagittal PD (**A**) and sagittal T2 FS (**B**) MR images demonstrate evidence for cartilage repair at the posterior weightbearing aspect of the lateral femoral condyle (arrowheads in **A** and **B**). The graft is appropriately positioned, and the overlying graft cartilage is intact and congruent with the adjacent native hyaline cartilage. There is minimal chondral fissuring at the anterior graft-cartilage interface (arrow in **A** and **B**). FS, fat saturation; MR, magnetic resonance; PD, proton density.

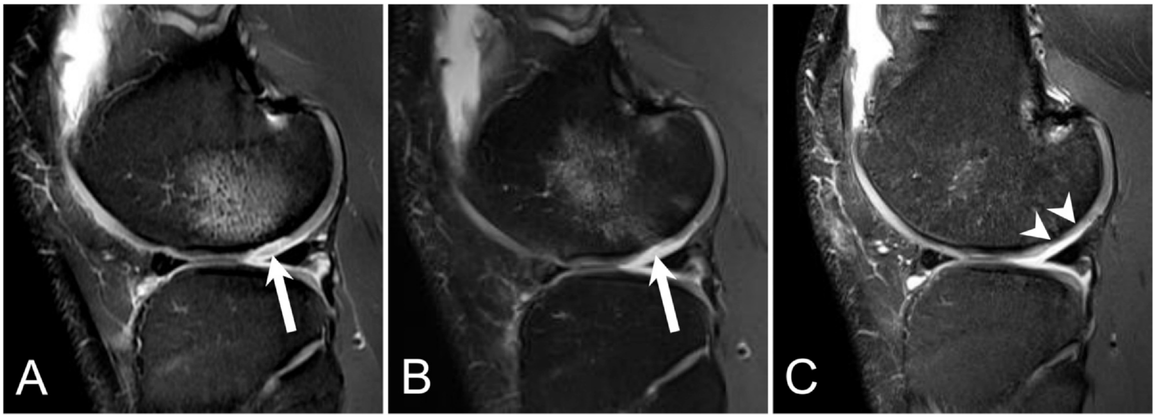

Fig. 19. Elite hockey player presents for MRI immediately following a noncontact knee injury. Sagittal PD FS MR image (**A**) through the lateral compartment demonstrates an acute osteochondral injury with a high-grade cartilage defect and adjacent delamination at the posterior weight-bearing surface of the lateral femoral condyle (arrow). Sagittal PD FS MR image (**B**) of the knee obtained 2 months later demonstrates interval improvement of the subchondral bone marrow edema but slight interval progression of the high-grade cartilage lesion (arrow). Sagittal PD fat-suppressed MR image (**C**) from an MRI performed 8 months after the initial injury demonstrates interval postsurgical changes of microfracture with moderate ingrowth of fibrocartilage (arrowheads) and interval resolution of the subchondral bone marrow edema. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging; PD, proton density.

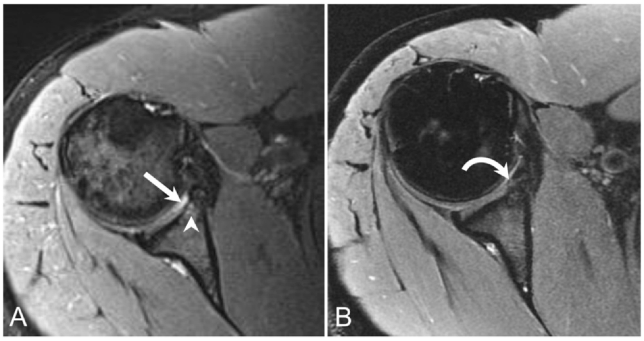

Fig. 20. Professional hockey player with chronic shoulder pain. Axial PD FS MR image (**A**) of the shoulder demonstrates focal full-thickness cartilage loss at the anteroinferior glenoid (arrow) with underlying subchondral edema/cystic changes (arrowhead). Axial PD FS MR image (**B**) from a follow-up MRI performed 1 year later, several months after intra-articular stem cell injection, demonstrates interval cartilage regeneration at the anteroinferior glenoid (curved arrow) with improvement of the accompanying subchondral bone marrow changes. FS, fat saturation; MR, magnetic resonance; MRI, magnetic resonance imaging; PD, proton density.

Although there is much interest in utilizing stem cell therapy for restoration of cartilage, there are inherent challenges, including the difficulty of carrying out long-term clinical trials, which limit its implementation.

After a repair procedure is performed, it is essential to evaluate the integrity of the repaired cartilage on follow-up imaging. The postoperative appearance of cartilage repair is dependent on the procedure performed and the timing of the MRI study relative to the procedure date, as it often takes months to years for the cartilage repair/restoration tissue to fully mature and integrate. As with the initial detection of articular cartilage injuries, MRI plays a critical role in the postoperative evaluation of surgical repair and other cartilage restoration procedures in the professional athlete. A comprehensive scoring system based on MRI, called "Magnetic Resonance Observation of Cartilage Repair Tissue" or MOCART knee score (and its most recent iteration, MOCART 2.0), has been shown to be highly reliable in evaluating the status of cartilage repair over time.[48] The MOCART 2.0 knee score evaluates the volume of cartilage defect filling, integration of repair tissue into adjacent native cartilage, surface and structure of the repair tissue, the signal intensity of the repair tissue, and any accompanying bony defect or bony overgrowth. MRI findings of inadequate or failed repair for the aforementioned techniques are well known and have been described in detail in the literature.[49–51]

Conclusion

This brief review covers many of the classic cartilage injury patterns in the professional athlete, utilizing case examples, and showcases the importance of MRI in both the initial detection and characterization of cartilage lesions, as well as the ensuing postoperative success of repair and/or restoration procedures in this unique patient population.

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Ethics approval

The article submitted is a narrative review and only includes de-identified case examples. No primary research or research subjects are included in the submission.

Declaration of Competing Interest

The authors declare no conflicts of interest.

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