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Ankle MR Arthrography: How, Why, When

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MR imaging has become established as the most effective imaging method in the assessment of numerous disorders of the ankle joint. MR arthrography extends the capabilities of conventional MR imaging because intra-articular injection of contrast solution allows selective examination of a joint, with controlled capsular distention and excellent depiction of the internal structures. There are some features of MR arthrography, however, that limit its clinical use, including the conversion of a noninvasive procedure into a mildly invasive one, exposing patients to ionizing radiation and the risks of intra-articular needle placement, and the increased cost and time required to perform MR arthrography compared with conventional MR imaging. Despite these limitations, MR arthrography is being used increasingly to evaluate intra-articular pathology of the ankle.

Indirect MR arthrography with intravenous administration of gadolinium also leads to an enhancement effect of the joint cavity, but it lacks capsular distention. This MR imaging technique is considered an alternative to direct MR arthrography in some cases.

This article reviews the role of MR arthrography in the evaluation of the ankle joint, considering techniques, pitfalls, complications, pertinent anatomy, and applications. It also provides a brief overview of the usefulness of indirect MR arthrography in this joint.

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How to perform ankle MR arthrography

MR arthrography of the ankle is a two-step procedure involving intra-articular injection of contrast solution before MR imaging. The injection can be performed in two main sites (Fig. 1A) at the anterior aspect of the ankle: immediately medial to the tibialis anterior tendon or medially to the tendon of the extensor hallucis longus [1–3]. The arthrogram usually is performed under fluoroscopy control; however, ultrasound, CT, or MR guidance also may be used [4–7].

The patient is placed in lateral decubitus position with the ankle in the lateral position and the front of the ankle facing the examiner. The course of the dorsalis pedis artery is palpated and marked to avoid its puncture. Using fluoroscopic guidance, a 23-gauge needle is inserted under sterile conditions into the tibiotalar joint medially to the tibialis anterior tendon with a slight cranial tilt to avoid the overhanging anterior margin of the tibia (Fig. 1B). Before the injection of contrast material, any fluid within the joint is aspirated so as not to dilute the contrast material. Intra-articular needle placement is confirmed with an injection of a drop of 2 mL of iodinated contrast material. If the needle is intra-articular, the contrast medium flows away from the needle tip and draws capsular recesses. Subsequently a mixture of 0.1 mL of gadolinium, 10 mL of saline solution, 5 mL of iodinated contrast material, and 5 mL of lidocaine 1% is injected until the joint capsule is properly distended (~10 mL). The presence of iodinated contrast material in the mixture ensures correct needle position and adequate capsular distention [8]. To prevent capsular disruption, contrast

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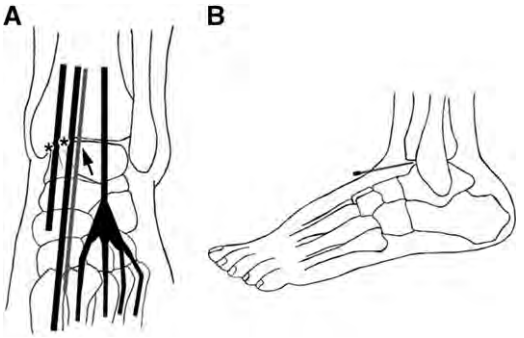

Fig. 1. Diagrams illustrate the injection sites for ankle joint MR arthrography. (*A*) Medially to anterior tibial tendon and medially to extensor hallucis longus tendon (*asterisk*). The course of the dorsalis pedis artery (*arrow*) should be avoided. (*B*) The needle is directed slightly cranially so that it can slide easily beneath the anterior lip of the tibia and advanced until its tip is seen between the distal tibia and the talus.

injection is stopped if the patient expresses discomfort or if high resistance is felt during the instillation of the solution. In a normal ankle, the injected contrast material forms an umbrella over the articular surface of the talus with prominent anterior and posterior capsular recesses. An upward extension of contrast material is seen between the distal tibia and fibula, filling the syndesmotic recess. In 25% of cases, contrast solution may enter the flexor hallucis longus (FHL) and flexor digitorum longus tendon sheaths and the subtalar joint [3,9,10]. There should be no tendon sheath filling on the lateral side of a normal ankle. After the injection, the needle is removed and the ankle is manipulated briefly to distribute the contrast medium uniformly.

Joint puncture can be performed in the MR imaging suite without radiologic guidance, using recognized anatomic landmarks and avoiding the need for iodinated contrast agents and ionizing radiation. It is particularly useful when there is limited access to a fluoroscopic suite [2]. The puncture point is located at the level of the anteromedial ankle joint, just medial to the tibialis anterior tendon, and about 5 mm proximal to the medial malleolus. After a brief learning period of fluoroscopic injection of the ankle joint, the injection can be performed easily without guidance with imaging methods by identifying anatomic landmarks.

Saline solution may be injected as the MR arthrographic contrast material. Normal saline is not an ideal contrast medium, however, because it has the same signal characteristics as preexisting joint effusion and para-articular fluid, and it is not possible to determine if they have occurred as a result of the saline injection [4–7].

Different studies have shown that patients who have undergone MR arthrography considered the discomfort less than expected [11,12]. No significant side effects have been reported that are attributable to intra-articular gadolinium solution [8,12]. The main complications of MR arthrography are joint pain that may last 1 to 3 days after joint puncture and vasovagal reactions. Articular distention in arthrography produces a feeling of pressure in the joint and pain on moving with variable intensity and progressive decrease in the first days after injection [12]. Vasovagal reactions may occur in patients undergoing arthrography, particularly in young athletic patients with low resting heart rates. Vasovagal reactions may be a result of coexisting circumstances, such as emotion, apprehension, and pain. Most patients who experience a vasovagal event have a quick recovery and are managed easily in the radiology suite. The routine administration of prophylactic atropine before ankle arthrography to block vasovagal reactions is unnecessary given the low incidence of these reactions (about 1% in the authors' experience). The number of vasovagal reactions diminishes considerably if the patient is not allowed to see the material of puncture and the procedure.

Joint infection is the major complication of arthrography; this extremely rare complication is independent of the type of substance being injected into the joint [12]. No reports exist on serious adverse events, such as anaphylactic shock or other events requiring treatment in an intensive care unit or hospitalization [12].

MR imaging should be performed shortly after intra-articular injection of contrast solution to minimize absorption of contrast and guarantee the desired capsular distention, although imaging delays of 2 hours are tolerated [3,7,12]. Imaging protocol should include axial, sagittal, and coronal planes with a dedicated extremity coil. Field of view may be decreased to optimize the visualization of intra-articular structures. T1-weighted spin-echo sequences with and without fat suppression maximize the signal intensity of contrast solution. Fat suppression is crucial in MR arthrography because fat and gadolinium have similar signal intensities on T1-weighted images, creating diagnostic difficulty. Fat suppression selectively decreases the signal from fat, while preserving the signal from contrast solution, confirming or excluding extra-articular contrast material. At least one sequence should be a fat-suppresed T2-weighted image for the detection of subtle bone marrow edema and extra-articular fluid collections

[2,3,5,7]. Several authors have postulated the use of oblique planes or forced positions of the foot in the assessment of the anterior talofibular (ATF) and calcaneofibular (CF) ligaments that follow an oblique course. The ATF ligament optimally is imaged with the patient's foot in plantar flexion on axial images or with the use of oblique axial images. The CF ligament optimally is imaged with the foot in dorsiflexion on axial images or with the use of oblique coronal images [13–16]. Three-dimensional gradient echo images allow reconstructions adapted to the anatomic course of these ligaments with good accuracy and render oblique planes or forced positions unnecessary. Three-dimensional gradient echo images also are helpful in the assessment of cartilage lesions and loose bodies. Overall, the choice of sequence depends on radiologist preference and sequence availability.

The most common pitfalls of MR arthrography of the ankle are extra-articular injection or leak of contrast material through the capsular puncture site, which can be confused with capsular disruption. Accumulation of contrast material in the anterior and posterior recesses of the tibiotalar joint, which manifests as smooth, encapsulated fluid outside the ligaments, can be interpreted as a ligamentous tear. The bulbous appearance of the posterior talofibular (PTF) ligament and posterior tibiofibular ligament on sagittal images can simulate loose bodies. This pitfall is avoided easily by the evaluation of consecutive sagittal images and knowledge of the location of these ligaments [17,18]. The pseudodefect of the talar dome is a normal groove at the posterior aspect of the talus. This defect should not be misinterpreted as an articular erosion or osteochondral defect [10,17,18]. The inadvertent use of undiluted gadolinium results in marked T1 and T2 shortening with fluid appearing low in signal. The instillation of air bubbles during injection may mimic loose bodies, although generally air bubbles rise to nondependent regions of the joint, whereas loose bodies gravitate to dependent locations.

Why and when to perform ankle MR arthrography

Ligamentous injuries

The ankle joint is supported by three ligamentous groups: the distal tibiofibular ligamentous or syndesmotic complex, the lateral collateral ligament (LCL) complex, and the deltoid ligament [19–23]. The LCL complex consists of three ligaments: ATF, CF, and PTF. The ATF ligament is located within the anterolateral joint capsule extending from the anteroinferior aspect of the lateral malleolus to the lateral talar neck (Fig. 2). The CF ligament is a cordlike structure that arises from the tip of the lateral malleolus and passes obliquely downward and posterior to insert at the posterolateral aspect of the calcaneus. It is extra-articular and closely associated with the inner sheath of the peroneal tendons. The PTF ligament is an intra-articular ligament that arises from the medial aspect of the distal fibula and passes almost horizontally to insert along the posterolateral tubercle of the talus [13–15,20,23,24].

The deltoid ligament or medial collateral ligament is composed of three superficial (tibionavicular, tibiospring, and tibiocalcaneal) and two deep (anterior and posterior tibiotalar) bands [17,20,22,25]. The deltoid ligament blends intimately with the tendon sheaths of the posterior tibial tendon and FHL and flexor digitorum longus tendons (Fig. 3). The anterior and posterior tibiofibular ligaments, the inferior transverse ligament, and the interosseous membrane form the distal tibiofibular syndesmosis ligamentous complex [17,20,22].

Approximately 85% of all ankle sprains are due to inversion forces and involve the LCL complex with a predictable sequence of injury involving first the ATF ligament, then the CF ligament, and finally the PTF ligament. Syndesmosis sprains are the second most prevalent (10%), and isolated medial sprains are third (5%) [17,20,26].

Most patients who had a lateral ankle sprain return to normal sport and daily living activity. Twenty

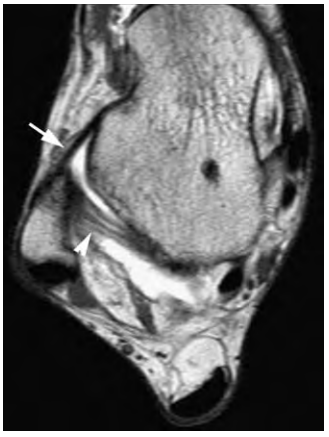

Fig. 2. Normal anatomy of the ATF and PTF ligaments of the LCL complex of the ankle. Axial T1-weighted MR arthrography shows the ATF ligament as a homogeneous low signal intensity structure (*arrow*) and the striated appearance of the PTF ligament (*arrowhead*) owing to the presence of fat interposed between its fascicles.

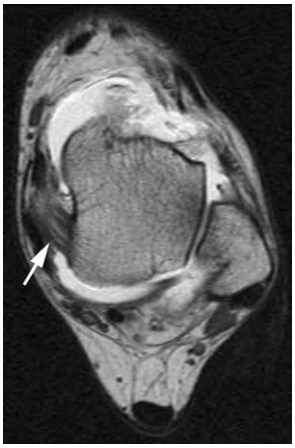

Fig. 3. Normal anatomy of the posterior tibiotalar fascicle of the deltoid ligament. Axial T1-weighted MR arthrography shows the normal striated posterior tibiotalar band of the deltoid ligament (*arrow*). Note the chronic tear of the ATF ligament.

percent to 40% of patients have residual pain, however, sufficient to limit or alter their activity [1,26–28]. These cases constitute a diagnostic and therapeutic problem. Chronic pain presenting after lateral ankle sprains may be secondary to a variety of reasons, such as ankle instability, ankle soft tissue impingement syndromes, sinus tarsi syndrome, peroneal tendon lesions, or osteochondral lesions of the talar dome [14,22,27,29].

Ankle instability can be characterized as mechanical or functional. Frequent giving way without evidence of anatomic ligamentous incompetency commonly is referred to as *functional instability,* whereas the objective finding of ligament incompetency (mobility beyond the physiologic range of motion) is termed *mechanical instability*. The incidence of functional instability after ankle sprains has been reported to range from 15% to 60% and seems to be independent of the degree of severity of the initial injury. Mechanical instability is much less prevalent. Chronic ankle instability often is characterized by repeated episodes of giving way with asymptomatic periods between episodes [22,28–31].

Deltoid ligament injuries most commonly occur in association with lateral ligamentous pathology, a fibular fracture, or syndesmotic injuries [17,20,25]. Isolated ruptures of the deltoid ligament are rare, but can occur as a consequence of an eversion-lateral rotation injury. Contusions and partial tears of the deltoid ligament, particularly of its posterior tibiotalar component, frequently are associated with inversion sprains, in which the deep posterior fibers of the medial deltoid ligament become crushed between the medial wall of the talus and the medial malleolus [20,32].

The incidence of syndesmosis sprains is probably higher than reported [19,25,28]. The mechanism of injury may be pronation and eversion of the foot combined with internal rotation of the tibia on a fixed foot. Syndesmosis injuries frequently are associated with eversion-type ankle fractures, particularly high fibular fractures (Maissoneuve) and rupture of the deltoid ligament. The presence of a syndesmosis sprain is a strong predictor for the likelihood of chronic ankle dysfunction [22,26,29].

Indications for the use of MR imaging may be limited to the evaluation of ligamentous injury in acute ankle injuries that show instability, stable acute injuries involving athletes or litigation, and repeated injuries or chronic ankle instability in patients in whom surgery is contemplated. MR imaging also has the advantage of depicting lesions often associated with ligamentous injuries, such as impingement syndromes, sinus tarsi syndrome, osteochondral lesions, and tendon tears [17,20,22].

Ankle ligaments are readily identified on MR images as low signal intensity structures joining adjacent bones usually delimited by contiguous high signal intensity fat. Heterogeneity and striation may be noted in some ligaments owing to the presence of fat interposed between their fascicles [15,17,22,24,33,34].

The MR imaging criteria for the diagnosis of acute tears of the ankle ligaments include morphologic and signal intensity alterations within the ligament (primary signs) and around it (secondary signs). Primary signs of ligament tear include discontinuity, detachment, nonvisualization, or thickening of the ligament associated with increased intraligamentous signal intensity on T2-weighted images indicative of edema or hemorrhage. Secondary signs of acute ligament injury include extravasation of joint fluid into the adjacent soft tissues, joint effusion, and bone bruises. Fluid within the peroneal tendon sheath can be a secondary sign of acute CF ligament injury. In chronic tears, secondary signs disappear, and the ligament can show thickening, thinning, elongation, and irregular or wavy contour [13,15,17,20,21]. Avulsion injuries are diagnosed easily in the acute and chronic settings showing the bone fragment adjacent to an irregular lateral or medial malleolus.

It has been shown in a cadaver study that the normal ligaments of the LCL complex and of the syndesmotic complex are shown better on MR arthrography than on conventional MR imaging [1–3,16]. The improved visualization resulted from the contrast

solution outlining more than one side of the ligament and because the ligaments were lifted away from the adjacent bone cortex.

MR arthrography is more sensitive and accurate than MR imaging in the evaluation of ligament tears [1–3,16]. The joint distention obtained with MR arthrography allows precise assessment of the thickness of the ligaments and their integrity at the insertion site, improving the diagnosis of acute and chronic tears (Fig. 4). Nonvisualization or extravasation of the contrast material anterior to the ATF ligament indicates tear of the ligament. A capacious anterior recess of the ankle joint allows the contrast agent to outline the anterior border of this capsular ligament, which represents capsular distention beyond the ligament and should not be confused with a tear. Disruption of the CF ligament often results in extravasation of contrast material lateral to the ligament and in communication of the ankle joint with the peroneal tendon sheath, which is attached to the superficial surface of the ligament (Fig. 5). Accumulation of contrast material in the peroneal tendon sheath at MR arthrography is an indirect but specific sign of CF ligament injury. Extravasation of contrast material into the soft tissues posterior to the PTF ligament indicates a tear of this ligament [1–3,16].

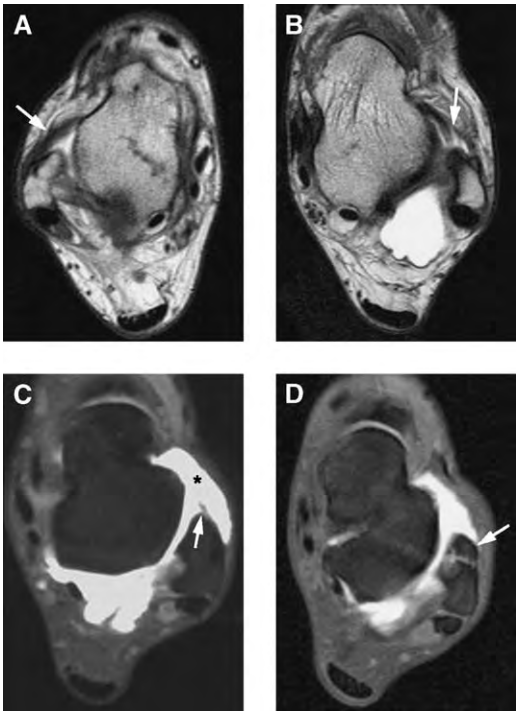

Fig. 4. Different appearances of chronic tears of the ATF ligament. (*A*) Axial T1-weighted MR arthrography shows irregular thickening and subtle waviness of the ATF ligament (*arrow*). (*B*) Axial T1-weighted MR arthrography shows focal disruption of the peroneal insertion of the ATF ligament (*arrow*). (*C*) Axial fat-suppressed T1-weighted MR arthrography shows disruption of the ATF ligament with small ligamentous end (*arrow*) and distention of the anterolateral recess (*asterisk*). (*D*) Axial T1-weighted MR arthrography reveals complete absence of the ATF ligament and avulsed osseous fragment (*arrow*) at the anteroinferior aspect of the lateral malleolus.

Lee et al [16] found no advantage to using MR arthrography when visualization of the deltoid ligament was required. In the authors' experience, MR arthrography with optimal articular distention outlines the deep component of this ligament and improves evaluation of partial tears of the fascicles of this component (Fig. 6). MR arthrography allows a better assessment of tears of the syndesmotic ligaments, manifested as thickening, lack of visualization, or irregularity of these ligaments, and is helpful in the detection of associated lesions (Fig. 7) [16].

Treatment of lateral ankle ligamentous injuries is conservative and includes the *RICE* regimen (*r*est, *i*ce, *c*ompression, *e*levation) and early controlled motion with functional brace. Surgical management of acute ankle sprains is rarely indicated. Numerous surgical techniques have been described to correct ankle instability with an 80% to 90% success rate. Direct repair of the ATF and CF ligaments has a success rate similar to that for augmented reconstructions (tenodesis) [1,22,28,29].

Treatment of the deltoid ligament is controversial and depends of the associated lesions. Grade I and II lesions are managed conservatively. Isolated acute deltoid tear (grade III injuries), avulsion of the medial malleolus, and chronic deltoid sprains with lengthening ligament are treated surgically [22].

Partial isolated syndesmosis tears should be treated conservatively. A complete tear is managed by suture of the ligament and temporary fixation of the tibia and fibula with a syndesmosis screw, cerclage, or Kirschner wires [22,29].

Ankle impingement syndromes

Ankle impingement syndromes are painful entities caused by the friction of joint tissues, which is the cause and the effect of altered joint biomechanics. The leading causes of impingement lesions are posttraumatic ankle injuries, usually ankle sprains [35–38].

From the anatomic and clinical viewpoints, these syndromes are classified as anterolateral, anterior, anteromedial, posteromedial, and posterior [35–38]. Patient history and an adequate physical examination

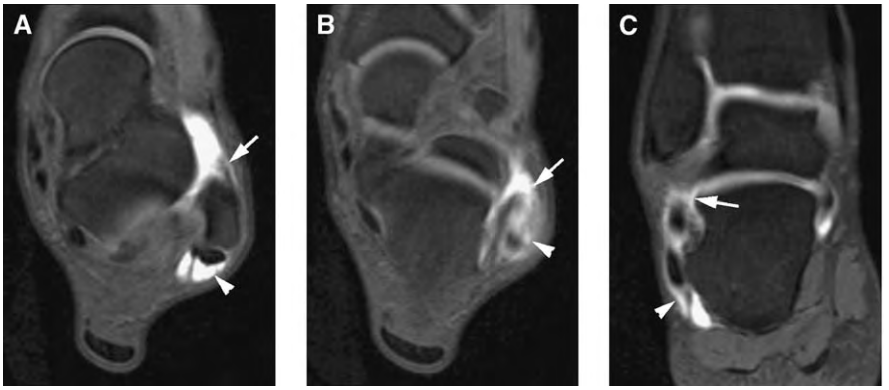

Fig. 5. Chronic tear of the ATF ligament and CF ligament. (*A* and *B*) Axial fat-suppressed T1-weighted MR arthrography shows disruption of the ATF (*arrow* in *A*) and CF ligaments (*arrow* in *B*). Note extravasation of fluid into the peroneal tendon sheath (indirect sign of the calcaneofibular ligament tear) (*arrowheads*). (*C*) Coronal fat-suppressed T1-weighted MR arthrography reveals discontinuity of the CF ligament (*arrow*) and communication of contrast with the peroneal tendon sheath (*arrowhead*).

can suggest a specific diagnosis in most cases. MR arthrography is the most useful imaging technique for detecting the soft tissue and osseous abnormalities present in these syndromes because it evaluates accurately the capsular recesses of the ankle [35–37].

Anterolateral impingement syndrome

Anterolateral impingement (ALI) is a relatively uncommon cause of chronic ankle pain produced by entrapment of abnormal soft tissue in the anterolateral gutter of the ankle [39–43]. It is estimated that approximately 3% of ankle sprains may lead to ALI. ALI is thought to occur subsequent to relatively minor trauma involving forced ankle plantar flexion and supination. Such trauma may result in tearing of the anterolateral soft tissues and ligaments without substantial associated mechanical instability. Repeated microtrauma and soft tissue hemorrhage can result in synovial scarring, inflammation, and hypertrophy in the anterolateral gutter of the ankle, with subsequent soft tissue impingement. Wolin coined the term *meniscoid* owing to its resemblance at surgery to meniscal tissue [35–43].

Other contributing factors include hypertrophy of the inferior portion of the anteroinferior tibiofibular ligament and osseous spurs. First described by Bassett et al [44], a separate distal fascicle of the anteroinferior tibiofibular ligament is a common variant. It becomes pathologic when a tear of the anterior talofibular ligament results in anterolateral joint laxity. With increasing joint laxity, the talus

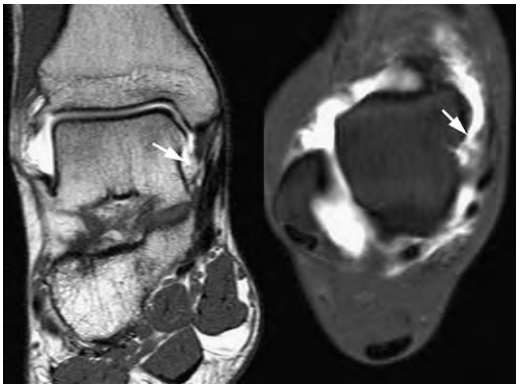

Fig. 6. Chronic tear of the deep component of the deltoid ligament. Coronal T1-weighted (*left*) and axial fat-suppressed T1-weighted (*right*) MR arthrography shows partial tear of the deltoid ligament involving the posterior tibiotalar fibers (*arrows*).

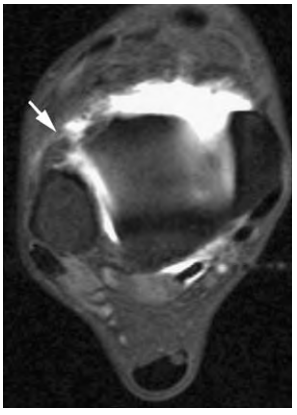

Fig. 7. Chronic syndesmosis sprain. Axial fat-suppressed T1-weighted image shows thickening and complete disruption of the inferior tibiofibular ligament (*arrow*).

extrudes anteriorly in dorsiflexion and comes into contact with the fascicle. Constant rubbing of the fascicle against the talus thickens the fascicle, which develops into an impinging lesion in the anterolateral gutter [44].

The clinical diagnosis of ALI can be established based on the combined presence of the following signs and symptoms: chronic ankle pain after an ankle sprain, anterolateral ankle joint tenderness, recurrent joint swelling, anterolateral pain with forced ankle dorsiflexion and eversion, pain during the single-leg squat, and lack of lateral ankle stability [37–39,42]. The clinical diagnosis of ALI is one of exclusion, however. Lesions producing similar symptoms have to be excluded before invasive treatment because similar symptoms can be attributed to peroneal tendon tears or subluxations, sinus tarsi syndrome, stress fractures, loose bodies, osteochondral lesions, bony impingement, and degenerative joint disease [35,37,38,41].

The presence of an abnormal soft tissue mass, hypointense on T1-weighted images and low or intermediate signal intensity on T2-weighted images, or a fibrous band in the anterolateral ankle gutter are MR imaging findings that suggest the diagnosis of ALI. The frayed margins of the torn ATF ligament should not be confused with the meniscoid lesion. Nevertheless, controversies exist regarding the accuracy of MR imaging in the diagnosis of ALI [39–42]. The evaluation of the anterolateral recess with conventional MR imaging is accurate only in the presence of substantial joint effusion [42].

MR arthrography is an accurate technique to assess the presence of soft tissue scarring in the anterolateral recess of the ankle and to determine its extent in patients with ALI before arthroscopy (Fig. 8). Robinson et al [41] in an arthroscopically controlled retrospective study found that MR arthrography assessment of the anterolateral soft tissues had an accuracy of 97%, sensitivity of 96%, and specificity of 100%. Accuracy was 100% with clinical anterolateral impingement. The absence of a recess of arthrographic fluid between the anterolateral soft tissues and the anterior surface of the fibula is another MR arthrography finding that suggests the diagnosis of ALI [41]. Nevertheless the identification of abnormal soft tissue itself does not imply the presence of clinical ALI. MR arthrography confirmation of anterolateral soft tissue abnormalities must be considered with the clinical findings [35,37].

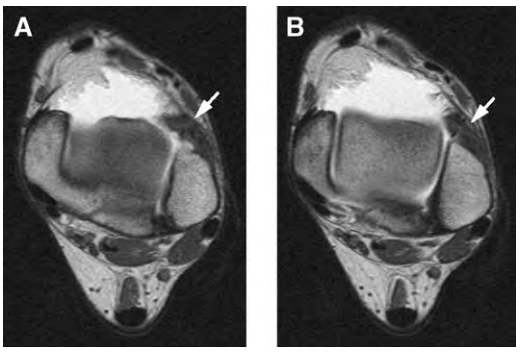

Fig. 8. Anterolateral impingement syndrome. (*A* and *B*) Serial axial T1-weighted MR arthrography reveals irregular soft tissue thickening in the anterolateral gutter (*arrows*).

Most patients with ALI respond to conservative therapy, including nonsteroidal anti-inflammatory drugs, rehabilitative physiotherapy, or local injection of steroids. If nonoperative treatment fails after 6 months, significant relief has been shown to be provided by arthroscopic débridement of hypertrophic synovial tissue in the anterolateral gutter [35–42].

Anterior impingement syndrome

Anterior impingement is seen more frequently in athletes subjected to repeated stress in dorsiflexion of the ankle, such as soccer players [35–37]. It is usually the result of impingement with trapping of soft tissues between a beaklike prominence at the anterior rim of the tibial plafond and a corresponding area over the apposing margin of the talus [35–37].

The cause and origin of anterior impingement are uncertain, and many factors are probably involved. It has been suggested that forced dorsiflexion results in repeated microtraumas on the tibia and talus leading to microfractures of trabecular bone or periosteal hemorrhage, which then heal with the formation of new bone. Another suggested mechanism in the etiology of these lesions is forced plantar flexion trauma, which causes capsular avulsion injury [35–37].

A classification for anterior ankle osteophytes has been developed, categorizing ankle spurs on the basis of spur size and the presence of associated arthritis. Lateral stress radiographs taken in maximum dorsiflexion may show physical impingement of the osteophytes [35,36]. MR arthrography is useful to assess the degree of cartilage damage, to delineate loose bodies, and to detect bone marrow edema and synovitis in the anterior capsular recess (Fig. 9) [35–37].

Conservative treatment, consisting of heel lifts, rest, modification of activities, and physical therapy, may be tried first. If there is persistent pain despite conservative treatment, arthroscopic or open resection of the spurs may be considered [35,36].

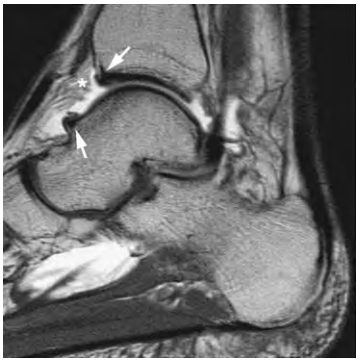

Fig. 9. Anterior impingement syndrome. Sagittal T1-weighted MR arthrography shows beaklike prominences at the anterior rim of the tibial plafond and over the opposed margin of the talus talar neck (*kissing lesion*) (*arrows*). Note synovitis in the anterior capsular recess of the tibiotalar joint (*asterisk*).

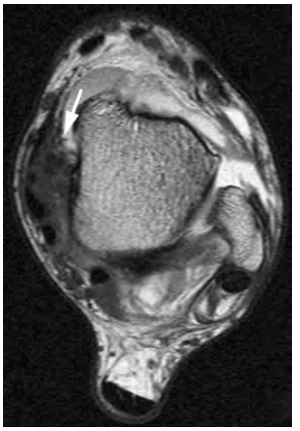

Fig. 10. Anteromedial impingement syndrome. Axial T1-weighted MR arthrography shows irregular soft tissue thickening in the anteromedial capsular recess (*arrow*) and thickening of the deep component of the deltoid ligament.

Anteromedial impingement syndrome

Anteromedial impingement is rarely an isolated condition, but is associated most commonly with an inversion mechanism of injury with lateral and medial ligamentous injury. It can be caused by a meniscoid lesion, represented by a mass of hyalinized connective tissue arising from a partially torn deep deltoid ligament or by a thickened anterior tibiotalar ligament. There also may be associated tibiotalar osteophytes. This thickened ligament or a meniscoid lesion, along with hypertrophic synovium, impinges on the anteromedial corner of the talus during dorsiflexion of the ankle [35–38,45].

Conventional MR imaging is not effective in detecting anteromedial impingement but can show a partially torn deep deltoid ligament. MR arthrography is the imaging method of choice, clearly defining the medial meniscoid lesion (Fig. 10), the thickened anterior tibiotalar ligament, and chondral or osteochondral associated lesions [35,45]. If conservative treatment fails, débridement of the impinging lesion by arthroscopic methods yields good clinical results [35,45].

Posteromedial impingement syndrome

Posteromedial impingement is a rare cause of ankle pain. It can occur after a severe ankle-inversion injury with the deep posterior fibers of the deltoid ligament becoming crushed between the talus and the medial malleolus. Initially the symptoms that predominate are from the lateral ligament tear. Inadequate healing of the contused deep posterior deltoid ligament fibers may lead, however, to chronic inflammation and hypertrophic fibrosis and metaplasia. In these cases, the fibrotic scar tissue may impinge between the medial wall of the talus and the posterior margin of the medial malleolus [35,46].

MR arthrography may show the thickened soft tissues (Fig. 11) and subchondral contusions of this entity [35]. This lesion generally cannot be fully appreciated arthroscopically via anterior portals in a stable ankle and requires a high index of suspicion and careful examination for the diagnosis to be made clinically. Arthroscopic surgery, via a posterior portal, or limited open surgery excision of the lesion is successful in resolving the pain [35,46].

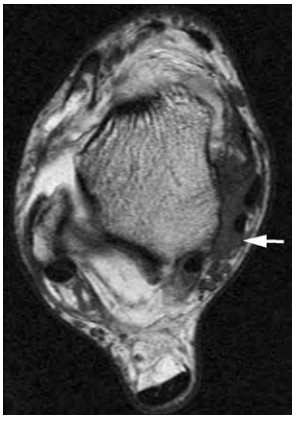

Fig. 11. Posteromedial impingement syndrome. Axial T1-weighted MR arthrography shows hypertrophic fibrotic tissue in the posteromedial aspect of the ankle (*arrow*).

Posterior impingement syndrome

Posterior ankle impingement (PAI) syndrome refers to a group of pathologic entities that result from repetitive or acute forced plantar flexion of the foot. [35,37,47] Ostrigonum syndrome, talar compression syndrome, and posterior block of the ankle are other names for the same syndrome. The mechanism of injury is the compression of the talus and the surrounding soft tissues between the tibia and the calcaneus, as a nut in a nutcracker, during plantar flexion of the foot. This syndrome is seen most commonly in classical ballet dancers, but it also can be seen in other sports [35,37,47,48].

The key factor in this syndrome is the anatomy of the posterior aspect of the ankle. The more common causes are the os trigonum (an accessory ossicle of the lateral tubercle that may persist unfused into adulthood in 7% of individuals), an elongated lateral tubercle termed a *Stieda process,* a downward sloping posterior lip of the tibia, the prominent posterior process of the calcaneus, and loose bodies. Synovitis of the FHL tendon sheath, the posterior synovial recess of the subtalar and tibiotalar joints, and the posterior intermalleolar ligament are possible soft tissue causes of impingement [35].

The presentation of the PAI syndrome can be as soft tissue inflammation, osseous injuries, or a combination of both. Osseous injuries include fracture, fragmentation, and pseudarthrosis of the os trigonum or lateral talar tubercle. The soft tissue changes associated are posterior ankle and subtalar synovitis and FHL tenosynovitis [35,37,47,48].

The diagnosis of PAI syndrome is based primarily on the patient's clinical history and physical examination results and is supported by findings on radiographs, CT, and MR imaging [37]. MR arthrography is useful in the assessment of PAI syndrome (Fig. 12). Findings include abnormal signal intensity in the lateral talar tubercle or os trigonum, consistent with bone marrow edema, which is believed to be the result of bone impaction and represents bone contusions or occult fractures. Inflammatory changes and synovitis in the posterior ankle soft tissues also can be found. The combined presence of marrow edema and posterior ankle synovitis may suggest the diagnosis of PAI [35,37].

The intermalleolar ligament often is not visualized on conventional MR imaging. MR arthrography improves the visualization of the intermalleolar ligament, which can readily be separated from the surrounding PTF ligament and the transverse inferior tibiofibular ligament [35,48,49].

The treatment of PAI syndrome is initially conservative. If conservative treatment fails, surgical excision of the osseous fragments, with potential release of the FHL tendon, may be indicated [21,35,36].

Cartilage lesions

Chondral lesions are common in the ankle joint. Symptoms of chondral lesions are mostly nonspecific, and clinical diagnosis is usually difficult. MR imaging is the best imaging method for the assessment of the articular cartilage [3,6]. Different MR imaging pulse sequences, including spin echo, gradient echo, fat-suppressed, and magnetization transfer contrast, have been used to study the articular cartilage. The reported sensitivities and specificities of different sequences vary. The following MR imaging grading is most commonly used: In grade I lesions,

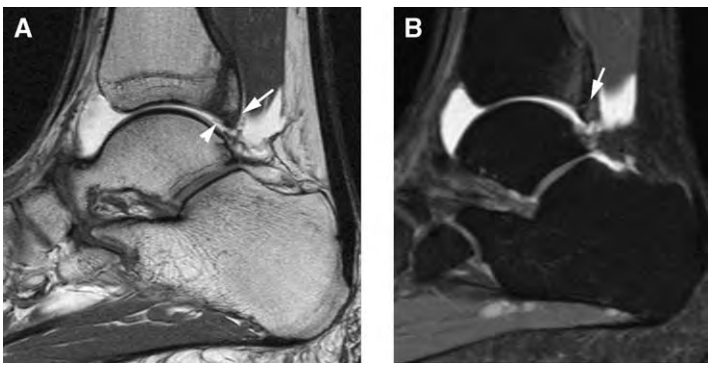

Fig. 12. Posterior impingement syndrome. (*A*) Sagittal T1-weighted MR arthrography shows a downward sloping deformity of the posterior lip of the tibia (*arrow*) after posterior malleolus fracture and focal chondral fraying (*arrowhead*). (*B*) Sagittal fat-suppressed, proton density–weighted spin-echo MR arthrography shows abnormal high signal intensity in the posterior malleolus (*arrow*).

MR images may show abnormal intrachondral signal with a smooth chondral surface and without alterations of the chondral thickness. Grade II lesions show mild surface irregularity with or without focal loss of less than 50% of the cartilage thickness. Severe surface irregularities with thinning of the cartilage thickness of more than 50% are present in grade III lesions. Grade IV lesions consist of complete loss of articular cartilage with denuded subchondral bone [3].

MR arthrography allows excellent delineation of the cartilage surface and provides good discrimination of higher grade cartilaginous lesions. MR arthrography can detect chondral lesions measuring 2 mm. Grade I chondral lesions have no surface abnormality and so may not be detected with MR arthrography [3,6,50].

Osteochondral lesions of the talus

Osteochondral lesion of the talus (OLT) is the accepted term for a variety of disorders including osteochondritis dissecans, osteochondral fracture, transchondral fracture, and talar dome fracture [51–53]. The primary lesional mechanism is a talar dome impaction owing to inversion injuries. These lesions typically involve medial or lateral aspects of the talar dome (Fig. 13). Medial lesions, which affect the posterior third of the talar border, are due to inversion injuries with plantar flexion of the foot and external rotation of the tibia (ie, impact between the posteromedial tibia and medial talar margin). Lateral lesions, which affect the anterior third of the talar border, are due to forced inversion and dorsiflexion

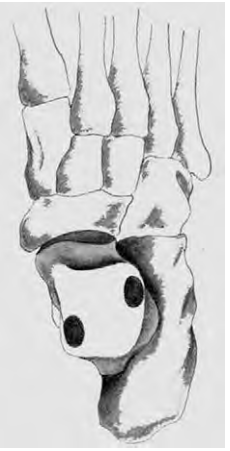

Fig. 13. Diagram shows the main locations of osteochondral lesions of the talus.

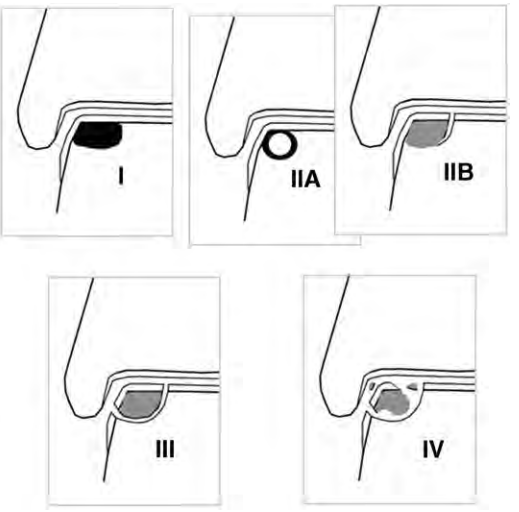

Fig. 14. Diagrams show classification of the osteochondral lesions of the talar dome.

of the foot (ie, impingement between the fibular styloid and the lateral margin of the talar dome). Medial and lateral aspects of the talar dome are involved in approximately 55% and 45% of the cases. Most lateral OLT appear thin and shallow, with the surface fragment greater in width than in depth. In contrast, medial OLT often have a deeper, crater-like appearance [36]. Clinical symptoms include exercise-related pain and less frequently sensations of clicking and catching and persistent swelling.

The classification introduced by Berndt and Harty is the most widely accepted staging system of osteochondral talar lesions [51–53]. This classification describes four stages depending on the integrity of the articular cartilage and the condition of the subchondral bone (Fig. 14). Stage I represents subchondral compression fracture, but the overlying articular cartilage remains intact. Stage II consists of a partially detached osteochondral fragment. In stage III, the osteochondral fragment is completely detached from the talus but is not displaced. In stage IV, the osteochondral fragment is detached and displaced, located away from the fracture site.

For a treatment decision, it is important to distinguish between stable and unstable lesions [19,34,53,54]. In stable OLT, including stage I and most stage II lesions, conservative treatment is recommended. Surgical treatment is advocated for unstable lesions, including stage IV and most stage III OLT. A subset of stage II lesions, especially laterally located lesions, may be treated surgically. Conversely, a subset of stage III lesions, in particular lesions located in the

medial talar border, may be managed conservatively [19,34,53,54].

MR imaging is effective in characterizing all stages of OLT, but is most useful in the identification of radiographically occult OLT and the stratification of in situ lesions into stable and unstable subsets. MR imaging diagnosis of OLT instability has relied on the interface between the osteochondral fragment and the parent bone on T2-weighted images. A stable or healed osteochondral fragment is characterized by the lack of high signal intensity at the interface between the lesion and the parent bone. The presence of a high signal line on T2-weighted images at the talar interface with the osteochondral fragment is the most reliable sign of instability. This high signal intensity may represent granulation tissue or fluid. An interface that is hyperintense, but not as much as fluid, indicates the presence of fibrovascular granulation tissue or developing fibrocartilage. At this stage, the lesion is unstable, but has the capacity to heal after a period of non–weight bearing or internal fixation [53]. If the interface is isointense with fluid or associated with cystic-appearing areas at the base of a nondisplaced lesion, surgery is indicated. There is controversy as to the accuracy of MR imaging in assessing the stability of the osteochondral fragment, with one author reporting a low accuracy of 50%, believed to be caused in part by the inability to distinguish fluid from granulation tissue on T2-weighted sequences. MR arthrography provides a better depiction of the talar chondral surface and is useful in differentiating a stage II from a stage III lesion by documenting intra-articular communication of fluid around the lesion (Fig. 15), which aids in planning therapeutic arthroscopy [2,3,5,6].

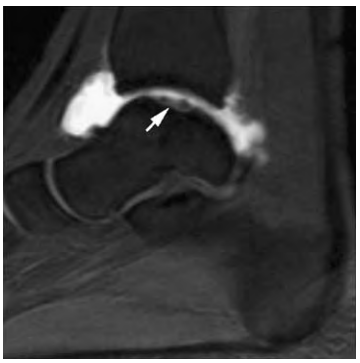

Fig. 15. Stage III osteochondral lesion. Sagittal T1-weighted MR arthrography shows a completely detached osteochondral fragment from the talus that remains located in the crater (*arrow*).

Surgical treatment consists of drilling of the lesion to improve perfusion or excision of the osteochondral fragment and débridement of the defect. Other surgical options include internal fixation of the fragment and bone grafting. In more advanced stage IV lesions, fragment excision and débridement of the defect is performed, whereas in earlier lesions (stage II and III), surgical treatment depends on the acute or chronic nature and size of the lesion [36,55,56].

Intra-articular loose bodies

Intra-articular loose bodies in the ankle joint may lead to impingement symptoms. Such bodies may consist of bone, cartilage, or both. Imaging is usually necessary to confirm the clinical diagnosis and to localize the intra-articular loose bodies before surgery. Radiographs are useful only when radiopaque intra-articular bodies are present. MR arthrography has been shown to be the best imaging technique for detecting osseous and cartilaginous loose bodies with a sensitivity of 86% and is significantly more sensitive than conventional MR imaging (Fig. 16) [57]. Air bubbles can mimic loose bodies at MR arthrography, but the distinction usually can be made by their nondependent position and typical appearance [2,5,6,7].

Synovial disorders

Synovial osteochondromatosis is an uncommon synovial metaplastic disorder that results in the formation of multiple ossified or calcified cartilaginous nodules [34,58,59]. It may arise in the synovium of joints, bursae, or tendon sheaths. Synovial osteochondromatosis is usually monarticular, with the knee involved most commonly. Clinical symptoms include pain, swelling, and locking of the affected joint. Radiographic findings depend on the presence of calcification or ossification within the nodules. Bone erosions are often seen. In one third of the cases, radiographs may be normal, owing to lack of ossification or calcification of the nodules. MR findings depend on the histologic composition of the nodules. Purely cartilaginous nodules are isointense with articular cartilage on all pulse sequences. Calcified nodules appear as signal void foci on all pulse sequences. By contrast, ossified nodules have a peripheral rim of low signal intensity on all pulse sequences and a central area of high T1 signal intensity corresponding to medullary fat [34,58,59]. MR arthrography in the absence of a significant joint effusion helps to confirm the diagnosis and to localize with precision the intra-articular bodies before surgery,

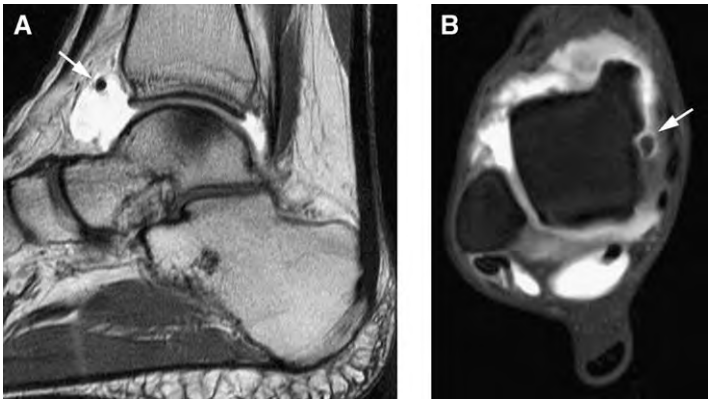

Fig. 16. Intra-articular loose bodies. (*A*) Sagittal T1-weighted MR arthrography shows osteochondral lesion of the talar dome and a small loose body in the anterior tibiotalar capsular recess (*arrow*). (*B*) Axial fat-suppressed T1-weighted MR arthrography shows a loose body in the anteromedial aspect of the ankle, anterior to the deep component of the deltoid ligament (*arrow*).

which is important because intra-articular bodies may be missed during arthroscopy (Fig. 17).

Pigmented villonodular synovitis (PVNS) is characterized by inflammatory proliferation of the synovium associated with deposits of hemosiderin [58]. It can be present in any joint, tendon sheath, or bursa but is seen most frequently in the knee, hip, ankle, and elbow. PVNS most often occurs in young to middle-aged adults and is more common in men. It may manifest as a focal mass or as a generalized lesion involving the entire joint space. Pressure erosions may be present in the diffuse form. These lesions manifest clinically as joint pain and swelling of long duration, and most are slowly progressive. PVNS has characteristic MR imaging features owing to the paramagnetic effect of hemosiderin, which produces areas of low signal intensity on T1-weighted and T2-weighted images [58]. Although hemosiderin typically is present, however, it is not invariably detected by MR imaging. Occasionally, hyperintense areas may be seen within the lesion on T1-weighted images owing to the presence of fat or synovial hemorrhage. Joint effusion is often present, producing hypointense areas on T1-weighted images and hyperintense areas on T2-weighted images [58].

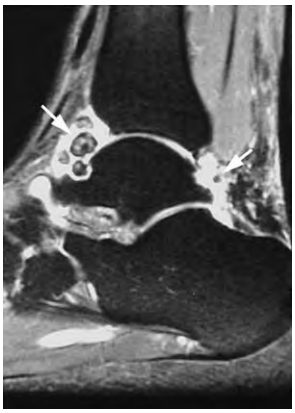

Fig. 17. Synovial osteochondromatosis. Sagittal fat-suppressed, proton density–weighted spin-echo MR arthrography shows multiple intra-articular osteochondral loose bodies (*arrows*).

MR arthrography shows accurately the location and extension of PVNS lesions before treatment. Arthroscopic synovectomy may be indicated for a focal mass or for an inactive form of diffuse disease. Combined partial arthroscopic synovectomy and low-dose radiation therapy and arthroscopic or open synovectomy are indicated in the treatment of diffuse PVNS.

Arthrofibrosis is an abnormal proliferation of fibrous tissue in and around a joint that can occur after ankle trauma and particularly after immobilization for an ankle sprain or fracture, causing persistent pain, limitation of ankle motion, and disability [60]. The adhesions that form often lead to stiffness and abnormal joint contact pressures and predispose the joint to cartilage degeneration [60]. MR arthrography is useful in the diagnosis of this entity, showing marked diminution in the volume of the capsular space, retracted and irregular margins of the capsular insertions, and adhesions or fibrous bands.

Indirect MR arthrography

Intravenous administration of a standard dose of gadolinium followed by 5 to 10 minutes of light

exercise can provide arthrogram-like images of the ankle joint [61–64]. This technique has been proposed as an alternative to direct MR arthrography. The main drawback of indirect MR arthrography is the lack of joint distention compared with direct MR arthrography. Another limitation of indirect MR arthrography is that juxta-articular structures, such as vessels, and the synovial membranes of bursae and tendon sheaths also show enhancement, which can lead to confusion with extravasation of contrast medium or the presence of abnormal joint recesses. Indirect MR arthrography may be useful in the evaluation of subtle cartilaginous defects, which are detected owing to enhancement of the cartilage defect and of the subchondral bone related to trabecular disruption and hyperemia [61–64].

In the assessment of OLT with indirect MR arthrography, contrast material enters the fragment-bone interface, indicating partial or complete detachment of the osteochondral fragment. Contrast can enter either from opacified synovial fluid or from the adjacent granulation tissue at the fragment-bone interface being hyperperfused. In either situation, the high T1 contrast at the interface is a sign of loosening (Fig. 18). Correlation of enhancement with T2-weighted signal improves accuracy [53,61–64].

Partial ligament tears may be identified by focal enhancement indicating hyperemia. Complete tears may be seen as enhanced joint fluid extending into the ligament defect (Fig. 19). Indirect MR arthrography also may be useful in the evaluation of ALI, outlining the inner aspect of the scarred ATF ligament [61–64].

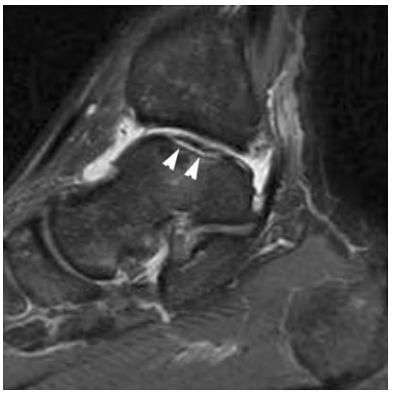

Fig. 18. Indirect MR ankle arthrography. Sagittal fat-suppressed T1-weighted MR arthrography shows contrast-enhanced fluid in the ankle joint around the osteochondral lesion of the talar dome (*arrowheads*), which indicates complete loosening of the osteochondral fragment.

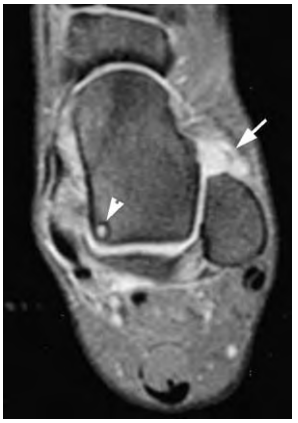

Fig. 19. Indirect MR ankle arthrography. Axial fat-suppressed T1-weighted MR arthrography shows contrast-enhanced fluid in the ankle joint that delineates a chronic tear of the ATF ligament (*arrow*) and small osteochondral lesion in the posteromedial aspect of the talar dome (*arrowhead*).

Indirect MR arthrography provides further assessment of extra-articular soft tissues of the ankle. Enhancement of extra-articular structures can highlight focal pathology, whereas lack of abnormal enhancement invariably indicates absence of disease in the region of interest. Enhancement about the plantar fascia is observed in patients with plantar fasciitis. With indirect MR arthrography, there is enhancement of fluid within the tendon sheath in the presence of tenosynovitis. Synovitis in the region of the tarsal tunnel is identified with enhancement around the posterior tibial nerve. Focal enhancement in the region of the sinus tarsi with indirect MR arthrography increases specificity of sinus tarsi pathology [61–64]. The overall advantage of indirect MR arthrography lies in gathering combined intra-articular and physiologic information.

Summary

MR arthrography has become an important tool for the assessment of a variety of ankle disorders. MR arthrography may facilitate the evaluation of patients with suspected intra-articular pathology in whom conventional MR imaging is not sufficient for an adequate diagnosis and be useful for therapy planning. MR arthrography is valuable in the evaluation of ligamentous injuries, impingement syndromes, cartilage lesions, OLT, loose bodies, and several

synovial joint disorders. Indirect MR arthrography is a useful adjunct to conventional MR imaging and may be preferable to direct MR arthrography in cases in which an invasive procedure is contraindicated or when fluoroscopy is not available.

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