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Ligamentous Injuries and the Risk of Associated Tissue Damage in Acute Ankle Sprains in Athletes

A Cross-sectional MRI Study

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Investigation performed at Aspetar, Qatar Orthopaedic and Sports Medicine Hospital, Doha, Qatar

Background: Ankle joint injuries are extremely common sports injuries, with the anterior talofibular ligament involved in the majority of ankle sprains. There have been only a few large magnetic resonance imaging (MRI) studies on associated structural injuries after ankle sprains.

Purpose: To describe the injury pattern in athletes who were referred to MRI for the assessment of an acute ankle sprain and to assess the risk of associated traumatic tissue damage including lateral and syndesmotic ligament involvement.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: A total of 261 ankle MRI scans of athletes with acute ankle sprains were evaluated for: lateral and syndesmotic ligament injury; concomitant injuries to the deltoid and spring ligaments and sinus tarsi; peroneal, flexor, and extensor retinacula and tendons; traumatic and nontraumatic osteochondral and osseous changes; and joint effusion. Patients were on average 22.5 years old, and the average time from injury to MRI was 5.7 days. Six exclusive injury patterns were defined based on lateral and syndesmotic ligament involvement. The risk for associated injuries was assessed by logistic regression using ankles with no or only low-grade lateral ligament injuries and no syndesmotic ligament damage as the reference.

Results: With regard to the injury pattern, there were 103 ankles (39.5%) with complete anterior talofibular ligament disruption and no syndesmotic injury, and 53 ankles (20.3%) had a syndesmotic injury with or without lateral ligament damage. Acute osteochondral lesions of the lateral talar dome were seen in 20 ankles (7.7%). The percentage of chronic lateral osteochondral lesions was 1.1%. The risk for talar bone contusions increased more than 3-fold for ankles with complete lateral ligament ruptures (adjusted odds ratio [aOR], 3.43; 95% CI, 1.72-6.85) but not for ankles with syndesmotic involvement. The risk for associated deltoid ligament injuries increased for ankles with complete lateral ligament injuries (aOR, 4.04; 95% CI, 1.99-8.22) compared with patients with no or only low-grade lateral ligament injuries.

Conclusion: About 20% of athletes referred for MRI after suffering an acute ankle sprain had evidence of a syndesmotic injury regardless of lateral ligament involvement, while more than half had evidence of any lateral ligament injury without syndesmotic involvement. Concomitant talar osseous and deltoid ligament injuries are common.

Keywords: magnetic resonance imaging; ankle sprain; risk factors; syndesmosis; athletes; osteochondral lesions

Ankle joint injuries are among the most common sports injuries and are usually the result of inversion and adduction of the plantarflexed foot.^{1,20,35} The anterior talofibular ligament (ATFL), which is the weakest of the 3 lateral ankle ligaments, is involved in the majority of lateral ankle sprains (up to 85%); the calcaneofibular ligament (CFL) is

involved in 50% to 75% of such injuries, and the posterior talofibular ligament (PTFL) is involved in less than 10%.^{14,24,26} In addition to injuries of the lateral ligament complex, involvement of the tibiofibular (syndesmotic) ligaments is not rare. The incidence of syndesmotic injuries ranges from 1% to 18% of ankle sprains.¹⁵

Syndesmotic injuries can be a significant source of missed playing time, especially for soccer players.⁹ Other structural damage such as involvement of the medial ligament complex and tendon or osseous injuries occurs as a consequence of ankle sprains, but data on the prevalence

of these additional findings are rare, as magnetic resonance imaging (MRI) is not routinely applied for the assessment of ankle sprains.¹³ The current diagnostic gold standard for the diagnosis of lateral ankle ligament injuries is a delayed physical examination combined with plain radiography according to the Ottawa rules (ie, a validated questionnaire that helps define the need for radiography after ankle sprains) for exclusion of a fracture and the assessment of mortise alignment.^{12,23,26}

As a clinical examination only yields relatively low sensitivity and high specificity in suspected syndesmotic injuries, MRI is often performed in addition to radiography.⁵ Prior studies have documented the ability of MRI to visualize the ligaments of the distal tibiofibular syndesmosis with 93% to 100% sensitivity and 96% to 100% specificity.^{16,17,29} In athletes, a timely diagnosis, especially of a high-grade syndesmotic injury, seems important as untreated injuries may cause persistent instability of the ankle joint.^{19,32} Although some syndesmotic injuries may be diagnosed radiographically, most of these injuries will be missed because of the inability of radiography to detect them in a reliable fashion.³⁴ Syndesmotic injuries may be associated with secondary injuries such as osteochondral lesions, bone contusions, or other ligamentous lesions that can be diagnosed only by MRI.⁴

Other indications for performing MRI for ankle sprains are injuries in athletes at advanced competitive levels for whom primary ligamentous surgical repair is contemplated and in patients with a history of chronic ankle instability.^{10,11} To date, no large MRI-based studies are available that have analyzed in detail lateral ligament and syndesmotic involvement and the prevalence of additional structural damage in athletes referred for MRI after an acute ankle sprain.

The aim of this retrospective study was to describe the structural injury patterns in athletes referred for MRI for acute ankle sprains. We further wished to assess the risk of associated structural injuries based on the injury pattern, characterized by: lateral ligament and syndesmotic ligament status; the deltoid ligament complex; the sinus tarsi and spring ligament structures; the flexor, peroneal, and extensor retinacula and tendons; the bony structures of the tarsus; and joint effusion of the tibiotalar and talocalcaneal joints.

MATERIALS AND METHODS

Study Design and Inclusion

The local institutional review board (IRB) approved the study design and granted exempt status (Anti Doping

Lab Qatar, IRB No. EX2013000001). No written patient consent was required for this retrospective analysis. Included patients were registered athletes under the National Sports Medicine Program (NSMP) of the State of Qatar. The NSMP is a centralized organization that oversees the medical diagnosis and treatment of athletes registered in sports clubs in Qatar, with the large majority of these being soccer players. Patients are seen primarily at the club level by a sports medicine physician, or they are directly referred to a specialized secondary referral sports medicine center for injury assessment. All imaging under the NSMP is performed at the secondary referral center. Included were all NSMP patients referred for MRI after suffering an acute ankle sprain during training or competition between 24 hours and 30 days before the MRI examination. Reasons for referral were not uniform, but the primary reasons for MRI were suspected lateral ligament damage, syndesmotic injuries, or acute osteochondral damage.

We searched the hospital picture archiving and communication system (PACS) for ankle MRI scans of athletes in a 4-year period from January 1, 2009 until December 31, 2012. The search yielded 697 MRI scans of the ankle performed in athletes during this period. Referral forms were searched for the terms “acute ankle sprain,” “twisting injury,” “sprain,” “syndesmosis,” “lateral ligaments,” and “ligament tear.” We identified 297 MRI scans of 261 patients based on these criteria. If a patient had more than 1 MRI scan, only the baseline MRI scan was included, which left 261 MRI scans for inclusion.

MRI Acquisition

All MRI scans were obtained with a 1.5-T large-bore MRI system (Espree, Siemens Healthcare, Erlangen, Germany), with a circumferential 8-channel extremity coil, using fat-saturated, turbo spin echo, proton density-weighted sequences in the sagittal (repetition time [TR], 2330 ms; echo time [TE], 32 ms; 3-mm slice thickness; 0.6-mm interslice gap; 22 slices; 320 × 224-pixel matrix; 2 excitations [NEX]; 15.9-cm² field of view [FOV]; echo train length [ETL], 7), coronal (TR, 2860 ms; TE, 32 ms; 3-mm slice thickness; 0.8-mm interslice gap; 27 slices; 320 × 224-pixel matrix; 2 NEX; 14.0-cm² FOV; ETL, 7), and axial (TR, 2990 ms; TE, 35 ms; 4-mm slice thickness; 0.8-mm interslice gap; 26 slices; 320 × 224-pixel matrix; 2 NEX; 14.0-cm² FOV; ETL, 7) planes. In addition, sagittal (TR, 493 ms; TE, 14 ms; 3-mm slice thickness; 0.6-mm interslice gap; 22 slices; 320 × 224-pixel matrix; 1 NEX; 15.9-cm² FOV;

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ETL, 1) and axial T1-weighted sequences (TR, 583 ms; TE, 14 ms; 4-mm slice thickness; 0.8-mm interslice gap; 26 slices; 320 × 224-pixel matrix; 1 NEX; 14.0-cm² FOV; ETL, 1) were acquired.

MRI Interpretation

The MRI scans were read by a single musculoskeletal radiologist (F.W.R.), with 15 years of experience in grading musculoskeletal MRI scans in a research context, on a high-resolution work station using eFilm software (eFilm workstation v 3.4, Merge Healthcare, Chicago, Illinois, USA). The MRI scans were read blinded for referral and clinical reports. Interobserver and intraobserver reliability was assessed with 30 randomly chosen MRI scans after a 4-hour calibration session using a different set of 20 MRI scans that were assessed and discussed in consensus. Interobserver reliability readings were performed by a second experienced musculoskeletal radiologist (A.G.) with 22 years of experience in standardized semiquantitative MRI assessment. Intraobserver reliability was tested after an interval of 6 weeks to avoid recognition bias.

The following structures were assessed using consensus definitions that were developed based on the existing literature and during calibration between the 2 readers as described above:

- The lateral ankle ligaments were graded as normal (grade 0), as a low-grade sprain (grade 1 = periligamentous high signal/edema on proton density-weighted sequences and no discontinuity of fibers), as partial disruption (grade 2 = partial discontinuity but preserved remnant fibers), as complete disruption (grade 3 = complete discontinuity), and as scar tissue (grade 4 = thinned or thickened ligament without discontinuity or periligamentous edema).^{18,22} The ATFL, CFL, and PTFL were assessed separately.
- The syndesmotic ligaments were assessed in a similar fashion from 0 to 4. The anterior-inferior tibiofibular ligament, the posterior-inferior tibiofibular ligament, the transverse tibiofibular ligament, and the interosseous membrane were assessed separately.

The following structures were assessed for associated injuries:

- Deltoid and tibiospring ligaments (the latter scored together with the superficial deltoid) (0-4 scale, scored separately for superficial and deep portions of the deltoid ligament complex): If any one of the bundles of the deep deltoid showed a lesion, the ligament was considered to be pathological (Figure 1). Deltoid contusions were scored in an identical fashion from 0 to 4 based on the ligamentous imaging appearance.
- Spring ligament complex (0-4 scale, scored separately for inferopltar longitudinal, mediopltar oblique, and superomedial).
- Sinus tarsi ligaments (0-4 scale, scored separately for the interosseous talocalcaneal and cervical ligaments).
- Peroneal, flexor, and extensor retinacula and tendons (0-3 scale; grade 0 = normal signal and morphology;



Figure 1. Deltoid ligament injury. Coronal proton density-weighted turbo spin echo magnetic resonance imaging shows complete disruption (grade 3) of the posterior tibiotalar ligament (arrow).

grade 1 = peritendinous edema, thickening, and intratendinous hyperintensities representing degeneration; grade 2 = intratendinous linear signal changes representing partial tears; grade 3 = complete disruption): Retinaculum changes were scored as grade 1 = surrounding edema, grade 2 = partial tears, and grade 3 = complete tears (Figure 2).

- Bone excluding the talus, that is, the fibula, tibia, calcaneus, navicular, and other (0 = normal, 1 = contusion, 2 = fracture).
- Talar osteochondral lesions (0 = normal, 1 = small contusion, 2 = large contusion, 3 = acute osteochondral lesion with intact cartilage, 4 = acute osteochondral lesion with cartilage injury, 5 = chronic osteochondral lesion): Small talar contusions were defined as being restricted to only 1 part of the talus, that is, the body, neck, or head. Large talar contusions were defined as involving at least 2 regions of the talus. Both definitions excluded contusions of the lateral talar dome adjacent to the subchondral plate, which were scored as an osteochondral lesion without surface damage (ie, as grade 3 lesions and not as grade 1 or 2 lesions).
- Acute osteochondral talar lesions were defined as areas of diffuse hyperintensity of the lateral talar dome directly adjacent to the subchondral plate with or without cartilage surface damage. A chronic osteochondral lesion was defined as a well-demarcated or partially cystic lesion in the same location with or without surrounding edema (Figure 3).⁷
- Effusion in the tibiotalar and talocalcaneal joints was scored separately, from 0 to 2, according to the amount

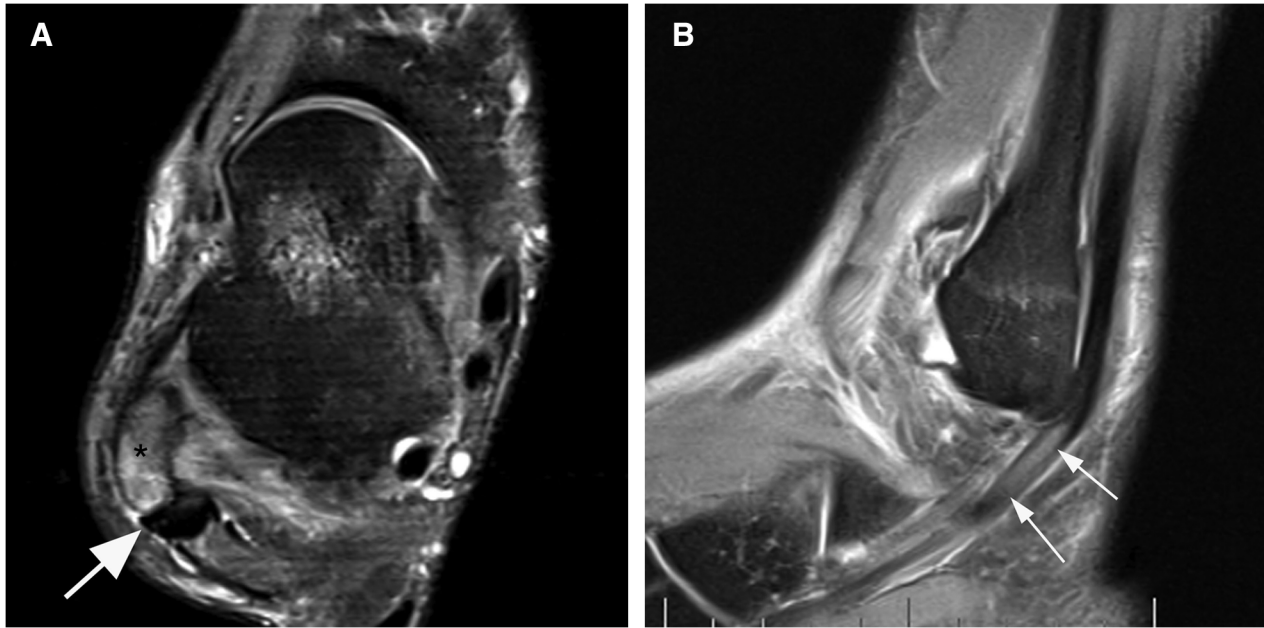


Figure 2. Tendon and retinaculum damage. (A) Axial proton density-weighted turbo spin echo magnetic resonance imaging (MRI) shows the beginning of subluxation of the peroneus brevis tendon laterally due to a grade 1 retinaculum injury with stripping of the superior peroneal retinaculum off the distal fibula (arrow). Note the fibular bone contusion (asterisk). (B) Sagittal proton density-weighted turbo spin echo MRI depicts a longitudinal split tear (arrows) of the peroneus brevis tendon, which was confirmed on T1-weighted axial MRI.

of capsular distension. Grade 0 represents only minimal physiological amounts of intra-articular joint fluid, grade 1 <50% of maximum capsular distension, and grade 2 \geq 50% of maximum capsular distension (Figure 4).

Analytic Approach

Six exclusive, different injury patterns were defined based on lateral and syndesmotomous ligament involvement: (1) no lateral ligament injury, no syndesmosis injury (noninjured ligaments were defined as grades 0, 1, or 4, assuming functional stability of scar tissue/grade 4 lesions); (2) low-grade ATFL and/or low-grade CFL and/or low-grade PTFL injury, no syndesmosis injury (ATFL = 1 or 2 and/or CFL = 1 or 2; and PTFL = 0, 1, 2, or 4); (3) complete ATFL injury, no CFL injury, no PTFL injury, no syndesmosis injury; (4) complete ATFL and partial/complete CFL injury and any grade PTFL injury, no syndesmosis injury; (5) partial/complete lateral ligament and syndesmosis injury; and (6) syndesmosis but no lateral ligament injury.

Descriptive statistics were applied to assess the frequencies of associated injuries based on these injury patterns. In addition, conditional logistic regression was performed to assess the risk of associated injuries in regard to the injury pattern. For this analysis, the injury patterns described above were combined: (1) ankles with no or only low-grade lateral ligament injuries and no syndesmotomous damage (patterns 1 and 2); (2) ankles with complete ATFL injuries but no syndesmotomous involvement (patterns

3 and 4); and (3) ankles with partial or complete syndesmotomous disruption (patterns 5 and 6). Ankles with no or only low-grade lateral ligament injuries and no syndesmotomous damage were used as the reference group. Results were adjusted for age and sex.

Reliability was assessed using weighted κ statistics and overall percentage agreement. The Fisher exact test was used to assess differences in the injury patterns based on age and sex.

All statistical calculations were performed using SAS software (v 9.3 for Windows, SAS Institute, Cary, North Carolina, USA). We considered a 2-tailed P value <.05 as statistically significant.

RESULTS

A total of 261 ankles of 261 patients were included. Patients were on average 22.5 ± 4.90 years old (range, 14-39 years). The majority were men ($n = 230$; 88.1%) and were registered with a soccer club ($n = 221$; 84.7%). The average time from injury to MRI was 5.7 ± 4.8 days (range, 1-26 days) for 214 patients. For 47 patients, the exact interval from trauma to MRI was not recorded, but recent trauma was verified by the referral form, which had to include the terms “acute” or “recent” and “trauma” or “sprain.”

In regard to the injury patterns, 105 ankles (40.2%) had no or low-grade lateral ligament injuries and no syndesmotomous damage, 103 (39.5%) had complete ATFL injuries (and any CFL or PTFL grade) and no syndesmotomous injuries,

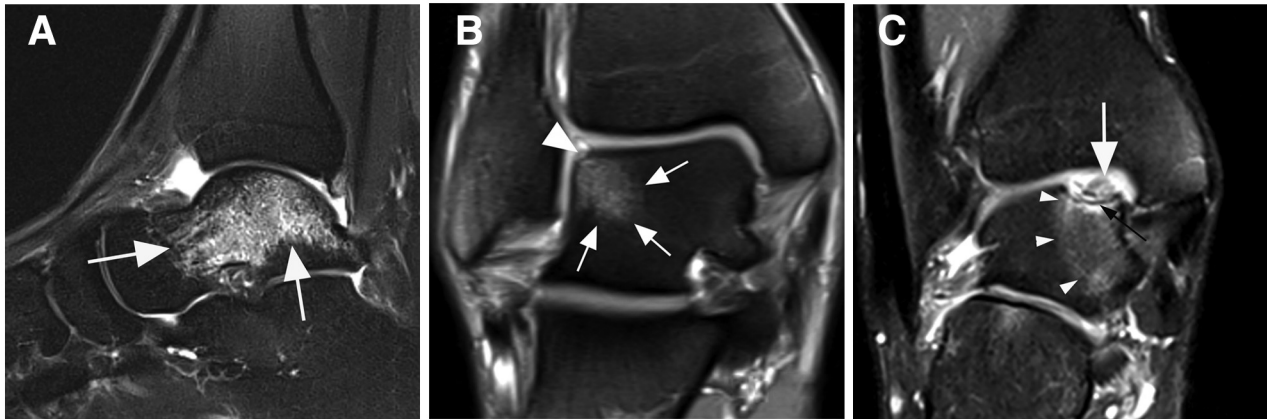


Figure 3. Bone injury on proton density-weighted magnetic resonance imaging. (A) A large talar bone contusion (grade 2) involving the talar body and talar neck is shown (arrows). No fracture line is seen. (B) Acute osteochondral injury to the lateral talar shoulder (grade 4). In addition to the bone contusion (arrows), a discrete acute surface lesion of the articular cartilage is shown (arrowhead). (C) Chronic osteochondral lesion of the talus (grade 5). An osteochondral fragment (white arrow) is unstable with a corresponding demarcated sclerotic rim at the talar dome (black arrow). There is also a large associated bone marrow lesion in the talar body (arrowheads).

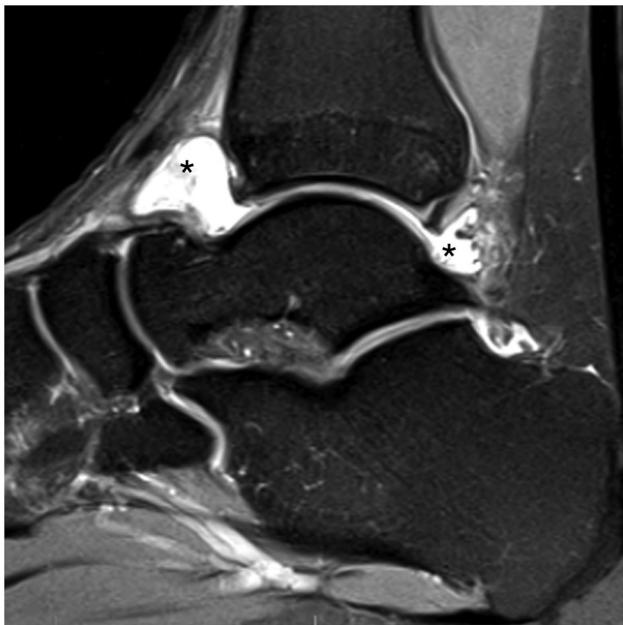


Figure 4. Traumatic joint effusion. Sagittal proton density-weighted fat-saturated magnetic resonance imaging shows a large amount of joint effusion anteriorly and posteriorly at the tibiotalar joint (asterisks).

and 53 ankles (20.3%) had syndesmotic injuries with or without lateral ligament damage. The distribution of the different injury patterns in regard to age and sex is detailed in Table 1. There were no statistical differences in frequencies of the injury patterns for age or sex.

Intraobserver reliability ranged from 0.67 (sinus tarsi) to 1.00 (retinacula, bone, and tendons). Interobserver

reliability ranged from 0.00 (retinacula) to 0.90 (syndesmosis). As some of the features were rare with regard to frequency, we also assessed percentage agreement, which ranged from 78.3% (effusion) to 100.0% (retinacula, bone, and tendons) for intraobserver reliability and from 68.3% (deltoid) to 98.9% (retinacula) for interobserver reliability. Detailed reliability results are presented in Table 2.

Several patients had additional spring ligament injuries (3.8%) and sinus tarsi involvement (16.1%). Retinaculum and tendon injuries were rare (Table 3). The risk for additional ligament (without deltoid) or tendon injuries did not increase for any of the injury patterns when compared with ankles without lateral or syndesmotic injuries as the reference (Table 4).

Ninety-two (35.2%) ankles had either partial or complete disruption (grade 2 or 3) of the deep, superficial, or both parts of the deltoid ligament complex. Including low-grade (grade 1) injuries, 128 (49.0%) ankles had suffered a deltoid ligament injury. The risk of partial deltoid tears markedly increased (adjusted odds ratio [aOR], 4.04; 95% confidence interval [CI], 1.99-8.22) for ankles with complete ATFL disruption and no syndesmotic injury and borderline increased for ankles with any syndesmotic injury (aOR, 2.24; 95% CI, 0.96-5.23). Appendix 1 (available online at <http://ajsm.sagepub.com/supplemental>) presents the data on deltoid ligament involvement in detail.

Additional osseous involvement ranged from 3.4% (navicular) to 23.0% (tibia) with no fractures but contusions only, which is detailed in Appendix 2 (available online). There was an increased risk for tibial osseous involvement for ankles with any syndesmotic injury (aOR, 4.46; 95% CI, 1.79-11.11), as depicted in Appendix 2.

Talar contusions not directly adjacent to the lateral talar dome were common, with 115 (44.1%) ankles showing small or large talar contusions. Acute osteochondral

TABLE 1
Injury Pattern According to Patient Age and Sex^a

Injury Pattern	No. of Ankles	Sex		Age, y					
		Male	Female	13-16	17-20	21-24	25-28	29-32	>32
No lateral ligament injury, no syndesmosis injury ^b	61	52 (85.2)	9 (14.8)	7 (11.5)	15 (24.6)	18 (29.5)	15 (24.6)	4 (6.6)	2 (3.3)
Low-grade ATFL injury, low-grade CFL injury, no syndesmosis injury ^c	44	38 (86.4)	6 (13.6)	4 (9.1)	10 (22.7)	8 (18.2)	13 (29.5)	7 (15.9)	2 (4.5)
Complete ATFL injury only, no CFL injury, no PTFL injury, no syndesmosis injury ^d	23	21 (91.3)	2 (8.7)	1 (4.3)	8 (34.8)	7 (30.4)	6 (26.1)	1 (4.3)	0 (0.0)
Complete ATFL injury and partial/complete CFL injury and any PTFL injury, no syndesmosis injury ^e	80	69 (86.3)	11 (13.8)	7 (8.8)	28 (35.0)	19 (23.8)	18 (22.5)	6 (7.5)	2 (2.5)
Partial/complete lateral ligament injury and syndesmosis injury ^f	32	30 (93.8)	2 (6.3)	2 (6.3)	9 (28.1)	11 (34.4)	5 (15.6)	3 (9.4)	2 (6.3)
Syndesmosis injury only, no lateral ligament injury ^g	21	20 (95.2)	1 (4.8)	4 (19.0)	8 (38.1)	4 (19.0)	3 (14.3)	2 (9.5)	0 (0.0)
Total	261	230 (88.1)	31 (11.9)	25 (9.6)	78 (29.9)	67 (25.7)	60 (23.0)	23 (8.8)	8 (3.1)
<i>P</i> value (Fisher exact test)		.81		.76					

^aValues are expressed as n (%). ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; PTFL, posterior talofibular ligament.

^bAll 3 lateral ligaments = 0, 1, or 4; all 4 syndesmosis ligaments = 0, 1, or 4. Grade 4 (scar) is treated as normal (equal to 0 or 1), assuming functional stability of scar lesions.

^cATFL = 1 or 2 and/or CFL = 1 or 2; and PTFL = 0, 1, 2, or 4; all 4 syndesmosis ligaments = 0, 1, or 4.

^dATFL = 3; CFL = 0, 1, or 4; PTFL = 0, 1, or 4; all 4 syndesmosis ligaments = 0, 1, or 4.

^eATFL = 3; CFL = 2 or 3; PTFL = 0, 1, 2, 3, or 4; all 4 syndesmosis ligaments = 0, 1, or 4.

^fATFL = 2 or 3; CFL = 2 or 3; PTFL = 0, 1, 2, 3, or 4; at least 1 of 4 syndesmosis ligaments = 2 or 3.

^gATFL = 0, 1, or 4; CFL = 0, 1, or 4; PTFL = 0, 1, or 4; at least 1 of 4 syndesmosis ligaments = 2 or 3.

TABLE 2
Intraobserver and Interobserver Reliability

Structure	Intraobserver Reliability		Interobserver Reliability	
	κ (95% CI)	Percentage Agreement	κ (95% CI)	Percentage Agreement
Lateral ligament complex ^a	0.88 (0.81 to 0.93)	80.0	0.85 (0.79 to 0.91)	76.7
Syndesmosis ^b	0.94 (0.88 to 0.99)	95.0	0.90 (0.81 to 0.99)	94.7
Deltoid ^c	0.81 (0.68 to 0.95)	86.7	0.41 (0.20 to 0.62)	68.3
Spring ^d	0.85 (0.70 to 0.99)	95.6	0.89 (0.76 to 1.00)	96.7
Sinus tarsi ^e	0.67 (0.32 to 1.00)	90.0	0.29 (-0.14 to 0.71)	80.0
Retinacula ^f	1.00 (1.00 to 1.00)	100.0	0.00 (0.00 to 0.00)	98.9
Bone, all ^g	1.00 (1.00 to 1.00)	100.0	0.83 (0.67 to 0.99)	86.7
Tendons ^h	1.00 (1.00 to 1.00)	100.0	0.71 (0.36 to 1.00)	97.5
Effusion ⁱ	0.74 (0.61 to 0.87)	78.3	0.72 (0.59 to 0.85)	76.7

^aAnterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament scored from 0-4.

^bAnterior-inferior tibiofibular, posterior-inferior tibiofibular, interosseous membrane, and transverse ligament scored from 0-4.

^cSuperficial/tibiospring and deep bundles of the deltoid ligament scored from 0-4.

^dInferoplantar longitudinal, medioplantar oblique, and superomedial scored from 0-4.

^eInterosseous calcaneal and cervical ligaments.

^fPeroneal, flexor, and extensor retinacula.

^gFibula, tibia, calcaneus, navicular, and other scored from 0-2; talar osseous lesions scored from 0-5.

^hPeroneal, flexor, and extensor tendons scored from 0-3.

ⁱTibiotalar and talocalcaneal joints scored from 0-2.

lesions of the lateral talar dome were seen in 20 (7.7%) ankles, of which 55% had intact cartilage and 45% showed cartilage damage. The percentage of chronic lateral osteochondral lesions was 1.1% (n = 3) (Appendix 3, available online). The risk for talar bone contusions increased for ankles with any syndesmosis injury but not for ankles with lateral ligament injuries (aOR, 3.43; 95% CI, 1.72-6.85) (Appendix 3).

DISCUSSION

In this large retrospective analysis of athletes with acute ankle sprains found on MRI, there was a prevalence of partial or complete lateral ligament disruption without syndesmosis involvement in 56% of patients and syndesmosis injuries with or without lateral ligament damage in about 20%. The most common findings with regard to associated

TABLE 3
Injury Pattern and Associated Ligament Injuries Without Deltoid Ligament Complex^a

Injury Pattern ^c	Associated Injuries: Ligaments/Tendons ^b				
	No. of Ankles	Spring Ligament Injury	Sinus Tarsi Involvement	Retinaculum Injury	Tendon Injury
No lateral ligament injury, no syndesmosis injury	61	4 (6.6)	14 (23.0)	1 (1.6)	0 (0.0)
Low-grade ATFL injury, low-grade CFL injury, no syndesmosis injury	44	3 (6.8)	4 (9.1)	1 (2.3)	1 (2.3)
Complete ATFL injury only, no CFL injury, no PTFL injury, no syndesmosis injury	23	0 (0.0)	3 (13.0)	1 (4.3)	3 (13.0)
Complete ATFL injury and partial/complete CFL injury and any PTFL injury, no syndesmosis injury	80	3 (3.8)	14 (17.5)	0 (0.0)	0 (0.0)
Partial/complete lateral ligament injury and syndesmosis injury	32	0 (0.0)	2 (6.3)	0 (0.0)	0 (0.0)
Syndesmosis injury only, no lateral ligament injury	21	0 (0.0)	1 (4.8)	0 (0.0)	0 (0.0)
Total	261	10 (3.8)	38 (14.6)	3 (1.1)	4 (1.5)

^aValues are expressed as n (%). ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; PTFL, posterior talofibular ligament.

^bAny lesion ≥ 1 .

^cSee Table 1.

TABLE 4
Risk of Associated Injuries Based on Injury Pattern: Ligaments/Tendons Without Deltoid Ligament Complex^a

Injury Pattern	Ankles (N = 261)	Associated Injuries: Ligaments/Tendons ^b			
		Spring Ligament Injury	Sinus Tarsi Involvement	Retinaculum Injury	Tendon Injury
No/low-grade lateral ligament injury, no syndesmotoc ligament damage ^c	105 (40.2)	7 (6.7)	18 (17.1)	2 (1.9)	1 (1.0)
aOR ^d		1.0 (reference)	1.0 (reference)	1.0 (reference)	1.0 (reference)
Complete ATFL injury, any CFL injury, any PTFL injury, no syndesmosis injury ^e	103 (39.5)	3 (2.9)	17 (16.5)	1 (1.0)	3 (2.9)
aOR (95% CI) ^d		0.42 (0.11-1.70)	1.03 (0.49-2.14)	0.45 (0.04-5.12)	3.17 (0.32-31.83)
P value		.23	.95	.52	.33
Syndesmosis injury and any lateral ligament injury ^f	53 (20.3)	0 (0.0)	3 (5.7)	0 (0.0)	0 (0.0)
aOR (95% CI) ^d		N/A	0.31 (0.09-1.12)	N/A	N/A
P value		N/A	.075	N/A	N/A

^aValues are expressed as n (%) unless otherwise indicated. ATFL, anterior talofibular ligament; aOR, adjusted odds ratio; CFL, calcaneofibular ligament; N/A, not applicable; PTFL, posterior talofibular ligament.

^bAny lesion ≥ 1 .

^cRows 1 and 2 combined from Table 3.

^dAdjusted for age and sex.

^eRows 3 and 4 combined from Table 3.

^fRows 5 and 6 combined from Table 3.

structural tissue injuries were bone contusions, especially in the talus (44%) and deltoid ligament (49%). Acute osteochondral lesions of the talus were seen in almost 8% of the athletes. The risk for tibial bone contusions increased more than 4-fold for ankles with any syndesmotoc injury but not for ankles with lateral ligament injuries. The risk for small talar bone contusions increased for ankles with any syndesmotoc injury but not for ankles with lateral ligament injuries.

Almost half of all ankle sprains occur during athletic activity, most commonly in basketball, football, soccer,

and running.³⁰ In a systematic review, the ankle was the most commonly injured body region in 24 of 70 sports included.⁶ Between 1% and 20% of these injuries involve the distal tibiofibular joint, which correlates with our finding of 20.3%.^{3,8,31} The most common trauma mechanism is inversion and adduction of the plantarflexed foot.³³ Ankle sprains can cause a significant financial burden, time lost to injury, and long-term disability.³⁶ In a recent review, van Rijn and colleagues²⁷ reported that 33% of patients had residual symptoms 1 year after an ankle sprain.

As MRI is not routinely performed in patients with acute ankle sprains, data on the prevalence of associated injuries are sparse. To our knowledge, the present study is the largest MRI-based evaluation to assess concomitant injuries in athletes with ankle sprains. One study of 90 ankles with severe sprains in nonathletes found acute or chronic syndesmotic injuries in 63% and acute or chronic ATFL damage in 74%; bone bruises or contusions were described in 24% of the ankles. Details on additional MRI findings of other structural injuries were not reported.⁴ In our study, we found acute bony involvement of the talus in 52% of the ankles and in other bones of the tarsus in 3% to 23%, with all of the latter representing bone bruises. Surprisingly, we did not find any fractures, which might be explained by the fact that most patients would have undergone a radiographic examination initially and those with fractures were not referred for MRI. The clinical prognosis of bone bruises is generally good, with normalization of the MRI appearance usually within 6 to 12 months after trauma.^{2,21,28}

Traumatic acute osteochondral lesions of the talus including subchondral bone contusions with or without articular surface damage were observed in 8% of ankles, while more diffuse bone contusions not exclusively located in the lateral talar dome were seen in 44%. Takao et al,²⁵ in a large arthroscopic MRI-based study, reported lateral talar osteochondral lesions in 71% of ankles with fractures and concluded that most lesions are traumatic in origin. Our cross-sectional design does not allow for an analysis of long-term outcomes of these lateral traumatic osteochondral lesions compared with more diffuse bone contusions at other locations of the talus.

Several limitations should be acknowledged. The retrospective nature of our study allowed us to describe the frequencies of lateral and syndesmotic ligament damage based on the injury pattern and to assess the risk of associated injuries. The purpose was not to associate these findings with clinical symptoms or functional status at the time of MRI. We do not know if the described MRI findings could have been diagnosed clinically but assume that associated injuries are more likely to be detected by MRI than clinically. Systematic, longitudinal clinical follow-up was not available, and for this reason, we do not know the relevance of the MRI findings in regard to return to activity. A further limitation of this study is that we could not compare our findings with a gold standard of surgery or histology. However, other studies using arthroscopic surgery have shown the high accuracy of MRI in detecting syndesmotic injuries.^{17,29} Also, our cohort consisted largely of male athletes who had quick and easy access to MRI, and extrapolating our data to a nonathletic population should be performed with caution.

Unfortunately, we were not able to specify the injury mechanism further, which would have been desirable in the interpretation of the different risks for associated structural damage. However, exact data on the injury mechanism beyond self-reports are difficult to gather unless video footage of the injury is available, which is rarely the case. We assume that the injury mechanism is likely one of the main drivers of damage severity in

patients with ankle sprains. The high prevalence of deltoid ligament damage has to be interpreted as a result of compression in most cases rather than traction unless a relevant rotatory component was involved. The clinical relevance of these lesions needs to be further investigated.

Patient inclusion was based on the status of being a registered athlete, with the large majority of these patients being soccer players. Unfortunately, we were not able to differentiate additional sports types further as this information was not available. Other sports commonly played and seen in the outpatient clinic for ankle sprains include track and field athletes and handball and basketball players. Reasons for referral for MRI were not defined in a standardized fashion, but most MRI scans were obtained to rule out or confirm lateral ligament and syndesmotic injuries based on injury mechanism, symptom presentation, and clinical examination. In a patient population of athletes, the threshold for prescribing an MRI examination certainly differs from that in the general population as the MRI outcome will have a different relevance for treatment choice, return to play, prognosis, and expectations of the athlete and coaching staff. However, our data do not support the usefulness of routine MRI in acute ankle sprains in a standard clinical setting.

In summary, in this population of athletes, we found injuries to the lateral ligament complex in about 70%, any syndesmotic involvement in about 20%, and acute osteochondral lesions of the talus in about 8%. As associated injuries, talar contusions were the most common finding. The role of talar contusions in regard to the development of posttraumatic chronic osteochondral lesions needs to be further assessed.

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