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Diagnostic and Therapeutical Challenges in the Lateral Ligamentous Complex Injuries of the Athlete's Ankle

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DIAGNOSTIC AND THERAPEUTICAL CHALLENGES IN THE LATERAL LIGAMENTOUS COMPLEX INJURIES OF THE ATHLETE'S ANKLE

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Thesis presented to obtain the Degree of Doctor in Medical Sciences
ACADEMIC YEAR 2019-2020

“Now this is not the end.
It’s not even the beginning of the end.
But it is perhaps, the end of the beginning…”

W. Churchill

ACKNOWLEDGMENTS

A very special thanks to:

Thesis Promotor Professor Jean-François Kaux,
for your inspirational and expert academic support

Thesis co-Promotor Professor Marc Martens
for being my Mentor in Sports Surgery

Thesis Committee Chair Professor Jean-Yves Reginster
for your academic leadership by example

Thesis Committee Members:
Professor Philippe Gillet
Professor Jean-Louis Croisier
Professor Olivier Bruyère
for the chance to learn from you and build with you

Professor Nebojsa Popovic
for being my Gold Medal coach in Sports Surgery

Professor Niek van Dijk
for changing my life in a way I could have never dreamt

Professor Kenneth Hunt
for opening your biomechanical lab and your valued friendship that developed through our collaboration

Professor Jan Ekstrand
for showing me the Power of Research in Orthopaedics and for your invigorating Sportsmedical Leadership

Professor Philippe Neyret
for the chance to join the International ISAKOS family and help build educational bridges in the domain of Foot and Ankle.

Professor James Calder
for teaching me your elite athlete world class expertise

Professor Guy Fabry UZ Leuven / Pellenberg Team
for believing in me and allowing me the entry to the Orthopaedic Speciality

Dr. Luc Van den Daelen, Dr. Paul Gunst and my valued colleagues at AZ Delta Roeselare, Belgium
for the chance to learn from the best

Dr. Alberto Grassi
for your statistical support in moments where I couldn't see the forest through the trees anymore.

Aspetar Hospital Senior Management Team and my valued Aspetar colleagues in Doha, Qatar
for the chance to develop myself and the teamwork that makes the dream work

Collaborative Universities:

University of Liège, Belgium
University of Colorado, USA
University of Pittsburgh, USA
Imperial College, London, UK
University of Boston, USA
UEFA elite Club Injury Study, EU
University of Bologna, Rizzolli Institute, Italy
University of Amsterdam, The Netherlands
University of Belgrade, Serbia
University of Paris, France
University of Rio de Janeiro, Brasil
for your valued collaboration and academic support

My Parents Anne-Marie and Michel
for simply being the best parents in the world

My Children Margaux, Estée, Alix and Charles
for making me feel such a proud dad

Valérie
for sharing love and life together Mi Vida

LIST OF ABBREVIATIONS

ADL	Activities of Daily Life
AP	Anteroposterior
ATFL	Anterior Talofibular Ligament
AiTFL	Anterior inferior Tibiofibular Ligament
AOFAS	American Orthopaedic Foot & Ankle (Score)
aOR	adjusted Odds Ratio
BI	Bias Index
CAI	Chronic Ankle Instability
CAIT	Cumberland Ankle Instability Tool
CFL	Calcaneofibular Ligament
CI	Confidence Interval
CM	Centimeter
COF	Center of Force
CT	Computer Tomography
3D	Dimensional
FAAM	Foot & Ankle Ability Measure
FAI	Functional Ankle Instability
FOV	Field of View
GRADE	Grading of Recommendations, Assessment, Development and Evaluation Guidelines
HO	Heterotopic Ossification
Hz	Hertz
ICC	Intraclass Correlation Coefficient
IOL	Interosseous Ligament
IOM	Interosseous Membrane
IQR	Interquartile Range
LAS	Lateral Ankle Sprains
LMM	Linear Mixed Models
MA	Moving Averages
MAI	Mechanical Ankle Instability
MCS	Medial Clear Space
MM	Millimeter
MPa	Intra-articular Tibiotalar Peak Pressure
MRI	Magnetic Resonance Imaging
N	Newton Force
NCCA	National Collegiate Athletic Association
NSMP	National Sports Medicine Program
OA	Osteoarthritis
OCD	Osteochondral defect

OLT	Osteochondral Lesion of the Talus
P	Prevalence
PTFL	Posterior Talofibular Ligament PiTFL Posterior inferior Tibiofibular Ligament
PTTL	Posterior Talotibial Ligament PROMS Patient Reported Outcome Measures
RCT	Randomized Controlled Trial
ROC	Receiver Operating Characteristic
RR	Relative Risk
ROM	Range of Motion
RTP	Return to Play
SD	Standard Deviation
SDC	Smallest Detectable Change
SE	Standard Error
SEM	Standard Error of Measurement
SF-36	Short Form 36-item Health Survey
3T	3 Tesla
TE	Echography Time
TC	Tibiocalcaneal
TN	Tibionavicular
TS	Tibiospring
TFCS	Tibiofibular Clear Space
TR	Repetition Time
TTFL	Transverse Tibiofibular Ligament
UEFA	European Football Association
VAS	Visual Analogue Scale

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PREFACE (ENGLISH VERSION)

When working with athletes from a medical point of view over the last 20 years, one can always see 4 main questions recurring in the athlete's mind after injury:

1. What do I have exactly?

2. What are the next steps for me in order to get back?

3. When can I return to play?

4. What can I do to prevent this in the future?

Although a lateral ankle ligament sprain is the most common injury in the world of sports, there's no evidence -based diagnostic or treatment algorithm available for several specific ankle sprain conditions.

The intention of this thesis is to dig into the complexity of the lateral ligament ankle injury and assist in finding answers to the related athlete's questions.

During the process of this academic work, new questions have already instigated future research projects and collaborations with experts in the field.

Therefore, I hope that the content of this thesis is not the beginning of the end, but more likely the end of the beginning…

PREFACE (VERSION FRANCAISE)

Ayant accompagné, sur le plan médical, des athlétes durant une période de vingt ans, j'ai remarqué que, après une lésion, ce sont toujours les mêmes importantes 4 questions qui surgissent dans l'esprit des athlètes.

1 –Quelle est la nature de ma lésion ?

2- Quelles sont les étapes à suivre pour revenir à mon niveau normal ?

3- Quand pourrai-je reprendre la pratique sportive ?

4- Que dois-je faire pour prévenir ce type de lésion à l'avenir ?

Quoique la lésion du ligament latéral de la cheville est une des lésions les plus fréquentes dans le monde du sport, il n'y a pas d'évidence, ni sur le plan diagnostic, ni sur le plan thérapeutique algorythmique, d'une attitude commune, adaptée à diverses conditions spécifiques.

L'Intention première de cette thèse est d'approfondir l'étude de la complexité du ligament latéral de la cheville, et de contribuer à fournir des réponses aux 4 questions précitées des athlètes.

Durant le processus de ce travail académique, des nouvelles questions ont déjà suscité des projets de recherches futures et de collaboration avec des experts dans la matière.

Voilà pourquoi, j'espère que cette thèse ne constituera pas le début de la fin, mais plutôt la fin du début.

INTRODUCTION

Lateral Ligament Ankle Injury

Introduction

Ankle injuries are reported to represent between 10% and 36% of all sport-related injuries [1,2]. Its incidence in sports has been reported between 0.324 and 9 per 1000 hours of activity [3,4], with variability most likely due to different definitions of injury and populations. Approximately 60% of the ankle injuries occur as a result of contact [5,6] and the overall ankle re-injury rate in sports is between 4% and 29% [5-7].
As a recent long-term study in professional football reported an ankle injury rate of 1/1000h [6], a professional 25-player squad club will suffer around seven ankle injuries in each season. In terms of time loss, an average of 16-24 calendar days are missed per ankle injury [3,6-8]. Severe injuries, i.e. more than 28 days absence, represent 10 to 17% of all ankle injuries [6,8,9].
However, up to 40% of the patients in the general population report residual symptoms after standard treatment for an acute ankle sprain [10,11], including chronic pain, recurrent instability and muscular weakness. The reported mean costs and economical burden per ankle sprain are € 360.60 ± 426.73 [12].

Functional Anatomy

The ankle joint can be regarded as a fork, in which tibia and both malleoli form a mortise to receive the talus. As a hinge joint, there is a single axis of movement that allows dorsiflexion (20°) and plantar flexion (50°). As the superior surface of the talus is narrower posteriorly, there is a looser fit within the fork during plantarflexion and most stability is then provided by ankle ligaments alone. This reduced intra-articular stability could potentially explain why most ligamentous injuries are sustained in plantar flexion [13].
The ankle joint is stabilized laterally by three ligaments: the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL) and the posterior talofibular ligament (PTF) [Fig.1]
.
The ATFL is a primary resistor of plantar flexion and internal rotation of the talus; it is the main stabilizer on the lateral side of the ankle joint [14] and it works in synergy with the CFL. This CFL is the primary restraint to varus tilting of

the talus within the mortise. Furthermore, the PTFL restricts external rotation when the ankle is in dorsiflexion [15].

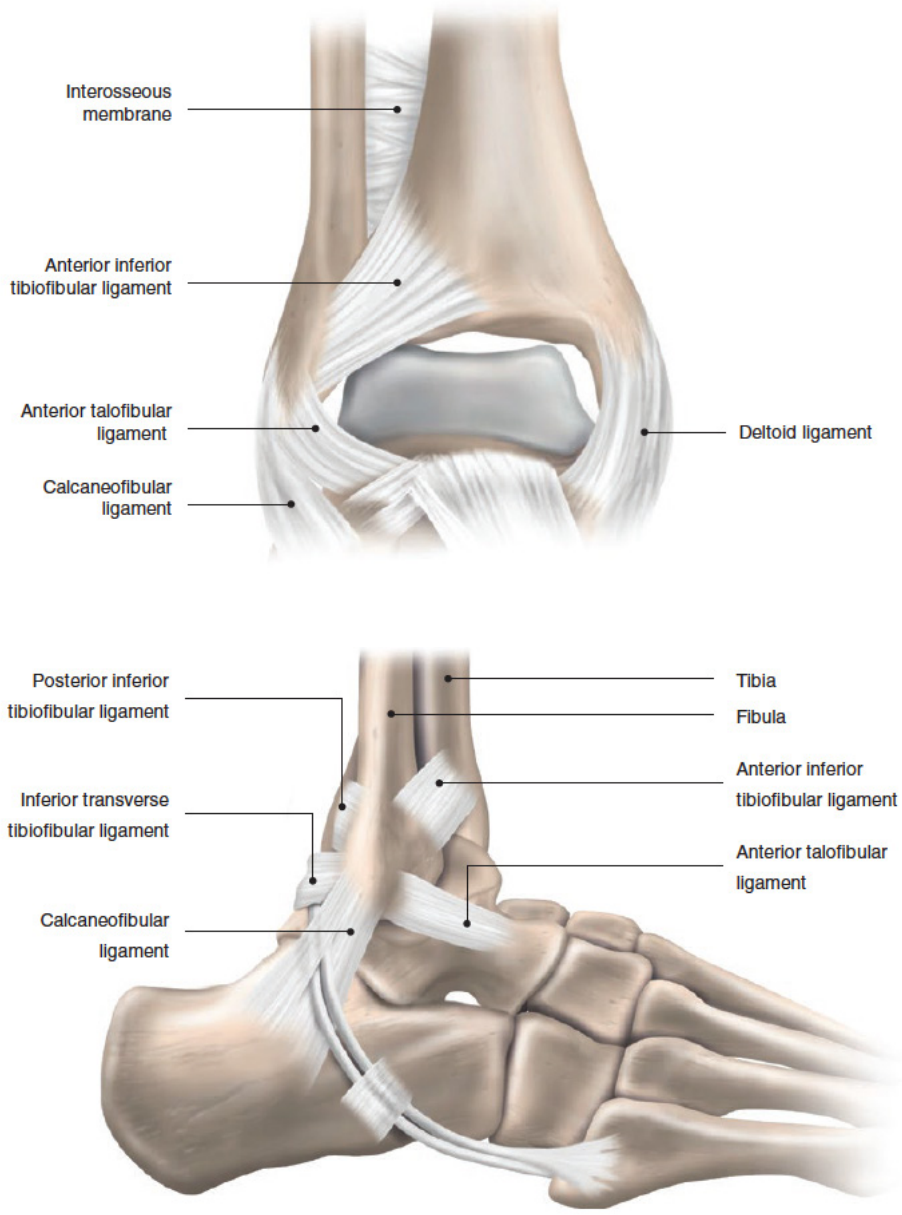

Figure 1. Antero-posterior and lateral view of the ankle ligaments

The tibiofibular syndesmosis or syndesmotic ligament complex consists of the anterior inferior tibiofibular ligament (AITFL), the posterior inferior tibiofibular ligament (PITFL) and the interosseous ligament (IOL) [Fig.2]. It ensures the stability between distal tibia and fibula and it resists the axial, rotational and translation forces which tend to distend the distal tibia and fibula [16,17]. The syndesmotic joint allows the tibia-fibula as a whole to adapt to the varying

width of the upper articular surface of the talus. This small amount of movement is important in normal walking and running.

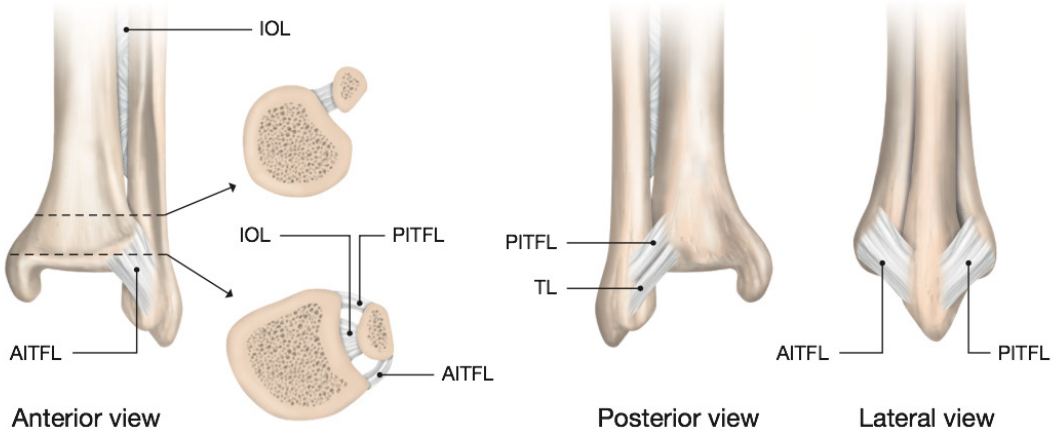

Figure 2. Detailed antero-posterior and lateral view of the ankle syndesmosis ligaments

Mechanism of Injury

The most common ankle sprain mechanism of injury is inversion of the plantar-flexed foot. A video analysis of ankle sprains in sports, revealed two common mechanisms that put the ankle in this vulnerable position: (1) impact by opponent on the medial aspect of the leg just before or at foot strike, resulting in a laterally directed force causing the player to land with the ankle in a vulnerable inverted position, and (2) forced plantar flexion when the injured player hits the opponent's foot when attempting to shoot or clear the ball [18]. As the ATFL is maximally stretched with inversion of the plantar-flexed foot and has the lowest tolerance to loads (approximately 150 N [19,20]), it is the first and often only ligament to sustain injury. As a result, the ATFL is the most frequently injured ligament of the ankle (90-95% [8,21]). When the mechanism of injury continues around the lateral aspect of the ankle, rupture of the ATFL is followed by damage to the CFL and finally to the PTFL.
Associated injuries include fractures, osteochondral lesions and damage to both the peroneus tendon and nerve. Chapter 2 offers a review that serves as a comprehensive analysis of the literature on ankle sprains, focusing on identification, treatment, and long-term outcomes of concomitant injuries in chronic ankle instability.
In Chapter 9, we present a review on why chronic ankle instability (CAI) resulting from an ankle sprain, might have severe long-lasting consequences on the ankle joint especially with the cartilage as primary victim.

Clinical presentation

Patients generally describe a sudden twisting of the ankle, inability to bear weight and localized pain and swelling. Patients with lateral ligamentous rupture report more immediate swelling and are more frequently compelled to stop their activities, than those without a rupture [22]. Ankle sprains may be accompanied by an audible snap or crack. All ligamentous and bony structures should be palpated for tenderness, including the whole length of the fibula and the base of the fifth metatarsal. If there is no pain on palpation of the ATFL, there is no lateral ligament rupture [23,24]. Note that approximately 40% of the patients with a lateral ligament rupture have pain on palpation of the medial malleolus, whereas 60% reports tenderness over the AITFL without rupture of this ligament, probably due to an anterior capsule tear [22]. The anterior drawer test evaluates ATFL laxity, whereas the talar tilt test aids in identifying CFL instability. The sensitivity to correctly diagnose an acute lateral ligament rupture during a delayed physical examination is 96 %, with a specificity of 84% [23,24].

Diagnosis

The Cumberland Ankle Instability Tool (CAIT) measures a subset of chronic ankle instability (CAI), namely functional ankle instability (FAI). In Chapter 6, we set out to translate and validate the CAIT in French because no French version existed.
The Ottawa ankle rules have been developed to help determine if X-rays are indicated after acute ankle injury. It is an accurate instrument to rule out fractures of the ankle, with a sensitivity of almost 100% [25]. Stress radiographs are generally not indicated in the routine diagnosis of lateral ligament injury, as they are difficult to perform and will not change the treatment protocol.
Ultrasonography and MRI can be useful in diagnosing associated injury (bone, chondral or tendon). Ultrasonography has been demonstrated to be an accurate investigation for ligamentous injury, but images may be difficult to interpret on retrospective review by other physicians. The sensitivity and specificity of ultrasonography for a lateral ligament rupture are 92% and 64%, respectively [24]. If ultrasonography is performed after an inconclusive delayed physical examination, sensitivity increases to 100% and specificity to 72% [25].

The sensitivity and specificity of MRI for ATFL injuries are 92-100% and 100%, respectively [26,27]. In comparison with arthroscopy, MR images correctly located the injured portion of the ATFL in 93%, whereas ultrasonography was

able to identify 63% [27]. Overall, MRI is the preferred imaging modality of choice for lateral ligamentous injury in the athlete. MRI is reliable in the diagnosis of lateral ligamentous ruptures and other associated injuries, including tendinous and syndesmotic tears, occult fractures and osteochondral lesions.
In Chapter 6, we present our additional findings that the presence of tibiotalar and talocalcaneal effusions on MRI is associated with an increased risk for these severe concomitant structural injury in acute ankle sprains.
In Chapter 7, we present our review of the literature and our study on MRI findings that links CAI with posterior ankle impingment. Our findings present a 10-fold higher need for posterior ankle arthroscopy in the professional athlete with os trigonum syndrome, in case of combined CAI.
In Chapter 2, we 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. We found that 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. We also found that concomitant talar osseous and deltoid ligament injuries are common.

Treatment

The majority of acute grade I-III lateral ligament injuries can be managed by non-operative measures [28] and is based upon the three stages of biological ligament healing: inflammatory phase, the proliferation phase (6 weeks to 3 months after trauma) and the remodelling or maturation phase (until 1 year post-injury). Most reviews comparing surgery versus conservative treatment for acute lateral ankle ligament injuries prefer functional treatment over surgical treatment [28,29]. However, surgical treatment may be beneficial on an individual basis in professional athletes [30]. An active rehabilitation protocol with the use of an ankle support was recently described in an evidence-based guideline [32].
Chapter 2 presents a comprehensive review on the different types of ankle sprains and arguments why there's no such thing as "a simple ankle sprain". The review furthermore highlights key points that need to be addressed before deciding for optimal treatment in CAI.
In Chapter 3, we present a biomechanical cadaver test that hypothesized that the CFL contributes considerably to lateral ankle instability. Our findings show that a higher grade sprains -that include CFL injury- result in significant decreases in rotation stiffness, peak torque, substantial alteration of contact mechanics at the

ankle joint, increased inversion of the talus and calcaneus, and increased medial displacement of the calcaneus.
Therefore, we conclude that repair of the CFL should be considered during lateral ligament reconstruction when injured, and there may be a role for early repair in high-Grade injuries to avoid intermediate and long-term consequences of a loose or incompetent CFL.
In an additional biomechanical cadaveric study on CFL (presented in Chapter 3), we conclude that restoring the CFL likely plays a relevant role in lateral ligament repair, however sufficient time for ligament healing should be allowed before rehabilitation stresses are applied.

Return to play (RTP) and Prevention

The time needed to RTP in lateral ligamentous injury depends on several factors, including severity of the injury, patient's ability and rehabilitation facilities available.
The current body of literature lacks formal criteria to assist in the decision to the football players' RTP with lateral ligament injury. In Chapter 8, we present a systematic review that identifies a clear deficiency in the literature pertaining to consistent, meaningful postoperative RTP timeline following lateral ankle ligament repair.
Our findings show that the published studies vary considerably in the metrics used for measuring patient-reported outcomes, and very few actually track them. Based on our conclusions, we recommend that further studies on outcomes following ankle ligament repair should include clear and consistent metrics for return to sport and level of play. Also, standardized and reproducible criteria for reporting RTP for athletes should improve the utility and applicability of outcomes data as surgical and rehabilitative techniques continue to advance.

Furthermore, in Chapter 8, we concluded that only very few articles describing outcomes of lateral ligament repair in athletes actually include return to play metrics. Considering the data available, athletes treated with open ankle ligament repair procedures returned to play almost 1 month earlier than athletes treated with arthroscopic procedures. As timing of return to activities is a valuable metric to compare surgical and rehabilitative techniques, we also concluded that more studies (that detail return to sport) are needed as part of a description of ankle ligament repairs.

Additionally, the primary victim of chronic ankle instability has been shown to be the cartilage. In Chapter 9, we describe the rehabilitation process for an ankle cartilage injury that requires a multidisciplinary and comprehensive approach. We joined the Pittsburgh international consensus derived from leaders in the field and believe this will assist clinicians with rehabilitation and return to sports after treatment of a cartilage injury of the ankle.

Syndesmotic Ligament Ankle Injury

Introduction

Injury to the syndesmotic ligaments, often referred to as a "high ankle sprain", occurs in 1-18% of patients with an ankle sprain, with some reports of injury in athletes of 3-6% [8,21]. It is likely that this is an underestimate, as 20% of athletes suffering an acute ankle sprain have evidence of syndesmotic injury on MRI [32]. Therefore, in Chapter 1, we determined the epidemiology of isolated syndesmotic injuries in +3500 professional elite football players. The incidence was seen to increase yearly over the past 15 consecutive seasons. We also found that the most common injury mechanism is by tackling and that the average return to play exceeded 5 weeks, which is substantially higher compared to a regular ankle sprain. Additionally, athletes with associated syndesmotic injury take twice as long to RTP compared to isolated lateral ligament sprains [33].

Functional Anatomy

The tibiofibular syndesmosis or syndesmotic ligament complex consists of the anterior inferior tibiofibular ligament (AITFL), the posterior inferior tibiofibular ligament (PITFL) and the interosseous ligament (IOL). It ensures the stability between distal tibia and fibula and it resists the axial, rotational and translation forces which tend to distend the distal tibia and fibula [16,17]. The syndesmotic joint allows the tibia-fibula as a whole to adapt to the varying width of the upper articular surface of the talus. This small amount of movement is important in normal walking and running.

Mechanism of Injury

The most accepted mechanism of injury for syndesmotic ankle sprains is a forceful external rotation of the foot and ankle with the ankle in dorsiflexion and the foot pronated [34]. Another injury mechanism for syndesmotic ankle

sprains is hyper-dorsiflexion. Forced dorsiflexion of the ankle causes the wider anterior talus to act as a wedge that can cause injury to the syndesmotic ligaments.

Clinical presentation

Specific symptoms, suggestive of syndesmotic injury are the inability to bear weight, swelling, pain during the push off phase of gait and pain anteriorly between distal tibia and fibula, as well as posteromedially at the level of the ankle joint [35]. The presence of 'high ankle pain', proximally up the anterolateral leg, is suggestive of a more significant injury and there is a significant correlation between how far tenderness extends up the leg and injury severity and time to return to sports [36]. Local tenderness is, however, not specific in the acute setting, as 40% of the patients with an ATFL disruption reported pain in the area of the AITFL, while arthroscopy showed no syndesmotic injury [24].
Numerous special tests are used to detect syndesmotic injury. The external rotation test and the squeeze test are most commonly described tests, but the Cotton test, the fibular-translation test and the cross-legged test have also been described. If clinical tests raise suspicion of a syndesmotic injury, additional imaging should be performed.
There are however no standardized criteria for the diagnosis and management of syndesmotic injuries, creating great ambiguity regarding optimal treatment. Traditionally, individuals with clinical and/or radiological suspicion of syndesmotic instability warrant an examination under anaesthesia and/or diagnostic arthroscopy to confirm and treat.

In Chapter 5, we present a new device that can identify clinical syndesmotic instability without the need of invasive arthroscopic procedures since the invasive process of diagnostic arthroscopy has inherent risks to the patient. We developed and validated this device that can dynamically evaluate the distal tibiofibular stability during external rotation of the ankle as an extension to the available clinical tests.

Diagnosis

Initial radiographs are recommended to assess bony integrity and stability of the ankle mortise. If there is a clinical or radiographical suspicion of a Maisonneuve fracture (i.e. pain in the region of the proximal fibula, painful swelling on the medial side without a fracture and isolated fractures of the

medial malleolus or malleus tertius), radiographs with full length views of the lower leg is needed. Biomechanical studies suggest that stress radiographs probably offer little advantage over plain views in assessing syndesmotic stability [37,38].
Computed tomography (CT) scanning is useful for detecting small avulsion fractures and is more accurate than radiographs in detecting subtle diastasis [39]. Recently, bilateral standing CT is emerging as an alternative diagnostic stress view, although prospective comparatively controlled data is lacking [40].

MRI effectively displays the structures of the syndesmosis and possible associated injuries, and is the investigation of choice for suspected syndesmotic ligament injury [37]. In comparison to arthroscopy, MRI shows a sensitivity of 100% and a specificity of 93% for AITFL injuries and sensitivity and specificity of 100% for PITFL tears [41]. It has demonstrated a high-degree of inter-observer agreement in identifying syndesmotic disruptions [42]. In a retrospective MRI study, a high prevalence of associated injuries was found, including osteochondral lesions (28%), bone contusions (24%) and osteoarthritis (10%) [43]. There are no reports that have assessed the association between extent of lesions on imaging and recovery time or clinical outcome.
In Chapter 6, we present our findings that the MRI-grading of the three major ligamentous complexes (according the Schneck grading system) and classification of syndesmosis injury (according the Sikka classification) resulted in reasonable to good diagnostic reliability. The reliability of the Schneck grading system for injury of the individual ligaments was however found to be limited.

Treatment

The classification of syndesmotic injury is divided into three grades: grade I represents a mild sprain to the AITFL without instability; grade II involves a tear of the AITFL and a partial tear of the IOL with some instability; and grade III represents definite instability with complete rupturing of all the syndesmotic ligaments [44].
Treatment is based on the severity of the syndesmotic injury. Grade I injuries without instability and only partial disruption of the AITFL, are treated with non-surgical management [45]. A 3-phase approach is recommended [46,47]; an acute phase, a subacute phase and an advanced training phase.
Treatment of grade II injuries depends on stability [37]. A recent study found that a positive squeeze test and injury to the ATFL and deltoid ligament are important factors in differentiating stable (type IIa) from dynamically unstable

grade II injuries (type IIb). For the athlete with a grade II injury and clinical or radiological suspicion of dynamic instability (type IIb), an examination under anaesthesia and arthroscopic visualization of the syndesmosis is recommended [48,49]. Dynamic diastasis of 2 mm or more warrants fixation [45]. Early anatomic reduction and fixation, leads to a potential quicker return to play in comparison with non-surgical treatment, although clinical data in athletes is lacking [50].
However, the diagnosis of isolated distal tibiofibular syndesmotic ankle instability proves to be a remaining challenge. Although diagnostic imaging has added value, it is limited in the detection of this distal syndesmotic ankle instability, especially in grade II injuries.
The gold standard remains intra-operative testing through arthroscopic probing while externally stressing the ankle in a sagittal direction. However, no validated arthroscopic guidelines have been established to distinguish a stable from an unstable syndesmotic ankle joint.
In Chapter 4, we present a cadaveric study that aims at providing biomechanical data that can assist the surgeon in the arthroscopic evaluation of syndesmotic injuries. The data from this study needs to be clinically correlated to ultimately assist in improving the outcome of patients with syndesmotic ankle injuries. This study offers to bridge the gap to the development of arthroscopic tools that can identify the need for surgical fixation to the syndesmosis based on the laxity of specific ankle ligaments that contribute to subtle instability.
Grade III injuries are uncommon in sports, inherently unstable and often associated with other injuries. Operative fixation is necessary to maintain anatomic reduction of the mortise. Screws or suture-buttons are used to stabilize the syndesmosis. Outcomes are similar, but the use of suture-button devices might lead to a quicker return to play and a lower rate of implant removal [51,52].
In Chapter 6, we evaluated static versus dynamic fixation of syndesmotic injuries. We found that dynamic fixation was able to reduce the number of complications and improve clinical outcomes compared to static screw fixation, especially malreduction and clinical instability or diastasis, at a follow-up of 2 years. A lower risk of re-operation with dynamic fixation was found compared to static fixation with a permanent screw.
Arthroscopic visualization can identify and address any additional intra-articular pathology. Furthermore, it can be used to confirm anatomic reduction of the syndesmosis [41]. In Chapter 6, we also present an evidence-based review of current techniques and implants for syndesmotic fixation.

Return to play and prevention

Athletes who sustain a syndesmotic ankle sprain typically experience much longer recovery periods than those who sustain a lateral ankle sprain [53]. RTP in grade I injuries is usually at 6-8 weeks post-injury, but is variable. Professional athletes with stable isolated grade II syndesmotic injuries are reported to RTP at a mean of 45 days, compared with 64 days for those with unstable grade II injuries [54]. In the case of surgically treated grade III injuries, the expected time frame to RTP is between 10 to 14 weeks [48,53], although RTP as early as 6 weeks has been described in case series [55].
In Chapter 8, we present our outcome series that establish the average time required to start with on field rehabilitation, team training and official match play in professional football players who were surgically treated for isolated unstable ankle joint distal syndesmosis injuries. We also identified 3 specific injury characteristics (a grade III injury, a combined cartilage lesion and young age) as predictors for a delayed return to match play.

Future studies

An ongoing international survey on the treatment of syndesmotic ankle injuries in athletes is depicted in Chapter 10.
We hope this study will not only show the geographical and specific therapeutical differences but will also instigate future research that can implement future validated guidelines on best practice.

And finally, Chapter 11 is the conclusion of it all: Teamwork in Medicine, Research and Education at the service of the Athlete's health.

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CHAPTER

1

EPIDEMIOLOGY OF SYNDESMOTIC INJURIES IN ELITE FOOTBALL

CHAPTER 1:
Epidemiology of syndesmotic injuries in elite Football

Epidemiology and return to play following isolated syndesmotic injuries of the ankle: a prospective cohort study of 3677 male professional footballers in the UEFA Elite Club Injury Study

British Journal of Sports Medicine (2017)

Lubberts B, D'Hooghe P, Bengtsson H, DiGiovanni C, Calder J, Ekstrand J

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Lubberts B, D'Hooghe P, Bengtsson H, DiGiovanni CW, Calder J, Ekstrand J.
Br J Sports Med. 2017 Dec 21. pii: bjsports-2017-097710. doi: 10.1136/bjsports-2017-097710.

ABSTRACT

Purpose:
The aim of the study was to determine the epidemiology of isolated syndesmotic injuries in professional football players.

Methods:
Data from 15 consecutive seasons of European professional football (UEFA Injury Study) between 2001 and 2016 contributed to the dataset of this study. Match-play and training data from a total of 3677 players from 61 teams across 17 countries have been included.
Team medical staff recorded player exposure and time loss injuries. Injury incidence was defined as the number of injuries per 1000 player-hours. Injury burden was defined as number of days absence per 1000 player-hours. Seasonal trends for isolated syndesmotic injury incidence, isolated syndesmotic injury proportion of ankle ligament injuries, and isolated syndesmotic injury burden were analyzed via linear regression.

Results:
The isolated syndesmotic injury incidence was 0.05 injuries per 1000 hours of exposure (95% CI 0.04 to 0.06) or 1 injury per team every 3 seasons. The injury incidence during match play was 13 times higher compared to during training, 0.21 (95% CI 0.16 to 0.26) and 0.02 (95% CI 0.01 to 0.02) respectively. Out of the 1320 ankle ligament injuries registered during the 15 seasons 94 (7%) were diagnosed as isolated syndesmotic injuries. An annual increase in injury incidence was observed (R^2=0.495, b=0.003, 95% CI 0.001 to 0.004, p=0.003). However, no significant annual change of injury burden was observed (R^2=0.033, b=0.032, 95% CI -0.073 to 0.138, p=0.520).
74% of the injuries were contact related and the mean (+/- SD) absence following an isolated syndesmotic injury was 39 (+/- 28) days.

Conclusion:

The incidence of isolated syndesmotic injuries in elite professional European football annually increased between 2001-16.

What are the new findings?

- Injury incidence during match play has increased over the past 15 seasons in elite European football
- Isolated syndesmotic injury in football is most commonly caused by tackling
- The average return to play after injury exceeds 5 weeks

How might these findings impact on our clinical practice?

- Our findings may assist in making football players, coaches, referees and the club medical staff aware of isolated syndesmotic injury and its consequences
- Our findings may contribute to the development of injury prevention strategies in football as they demonstrate that isolated syndesmotic injuries are most commonly caused by player-tackling

INTRODUCTION

Ankle syndesmosis injury may occur in many forms, commonly classified into isolated ankle syndesmosis injury or with an associated fibula fracture. An isolated injury may occur to any one of the three distinct ligaments (the anterior inferior tibiofibular ligament, the interosseous tibiofibular ligament, and the posterior inferior tibiofibular ligament),[1,2] but will most commonly involve the anterior inferior tibiofibular ligament.[3,4] The most common mechanisms of syndesmotic ligament injury are ankle external rotation and hyperdorsiflexion of the ankle, causing the talus to rotate in the mortise and the fibula to rotate externally and moving posteriorly and laterally, providing stress to the anterior inferior tibiofibular ligament.[3-5]

Isolated syndesmotic injuries occur more commonly in athletes than in the general population.[5-13] Certain sports are characterized by a higher proportion of ankle syndesmosis injuries; these include boot immobilized sports[5-8] such as skiing and ice-hockey, as well as collision sports such as American football, wrestling, and rugby.[9,10,12,14] For football, however, epidemiological data on isolated syndesmotic injuries is limited. Mauntel et al.[15] studied isolated syndesmotic injuries in 25 sports during six seasons and described the incidence rate, injury mechanism, recurrence, and time to return to activity of non-

professional football players. Due to differences in competition level, speed of the game, body shape of the players, and playing calendar it is expected that epidemiology and etiology of syndesmotic injuries differ between non-professional and professional players. A better understanding of how and when professional players incur these injuries may help the development of preventive strategies as well as providing important data regarding expected return to play times.[16]

In this study, we aimed to determine the injury incidence and epidemiology of isolated syndesmotic injuries of the ankle in professional football players over a 15 year period. In addition, we assessed the time to return to competition following an injury.

MATERIALS & METHODS

This is a substudy of a long-term prospective cohort study evaluating men's professional football in Europe since 2001.[17] The current study includes data from 15 consecutive seasons of European professional football between 2001 and 2016. During the study period, a total of 3677 players from 61 teams representing 17 countries have been included (table 1).

Table 1. Overview of amount of teams, exposure, and injuries per season

Season	Teams	Exp. Total (hours)	Exp. training (hours)	Exp. Match (hours)	Ankle ligament injuries	Ankle ligament injuries	Ankle ligament injuries	Syn. injuries (total)(ii)	Syn. injuries (training)	Syn. injuries (match)(ii)
01/02	11	69447	57915	11532	71 (1.02)	38 (0.66)	33 (2.86)	1 (0.01)	0 (0.00)	1 (0.09)
02/03	9	61777	51824	9954	41 (0.66)	12 (0.23)	29 (2.91)	1 (0.02)	0 (0.00)	1 (0.10)
03/04	11	64639	53866	10773	49 (0.76)	23 (0.43)	26 (2.41)	3 (0.05)	2 (0.04)	1 (0.09)
04/05	9	58257	48753	9504	44 (0.76)	18 (0.37)	26 (2.74)	2 (0.03)	0 (0.00)	2 (0.21)
05/06	17	102017	85446	16571	65 (0.64)	21 (0.25)	44 (2.66)	3 (0.03)	0 (0.00)	3 (0.18)
06/07	17	110658	93471	17187	89 (0.80)	43 (0.46)	46 (2.68)	3 (0.03)	3 (0.03)	0 (0.00)
07/08	14	95630	80294	15336	58 (0.61)	22 (0.27)	36 (2.35)	5 (0.05)	1 (0.01)	4 (0.26)
08/09	14	99181	83698	15483	77 (0.78)	31 (0.37)	46 (2.97)	3 (0.03)	1 (0.01)	2 (0.13)
09/10	18	123751	104534	19216	73 (0.59)	24 (0.23)	49 (2.55)	7 (0.06)	2 (0.02)	5 (0.26)
10/11	20	132314	110755	21559	83 (0.63)	38 (0.34)	45 (2.09)	5 (0.04)	2 (0.02)	3 (0.14)
11/12	31	213787	180742	33045	145 (0.68)	55 (0.30)	90 (2.72)	10 (0.05)	4 (0.02)	6 (0.18)
12/13	34	210069	176202	33868	162 (0.77)	54 (0.31)	108 (3.19)	11 (0.05)	2 (0.01)	9 (0.27)
13/14	39	257517	216619	40898	154 (0.60)	50 (0.23)	104 (2.54)	15 (0.06)	4 (0.02)	11 (0.27)
14/15	31	229372	195124	34247	118 (0.51)	36 (0.18)	82 (2.39)	17 (0.07)	4 (0.02)	13 (0.38)
15/16	29	208765	177506	31259	91 (0.44)	33 (0.19)	58 (1.86)	8 (0.04)	3 (0.02)	5 (0.16)

Exp; exposure, Syn; syndesmotic, ii; Injury incidence/1,000 exposure hours

Exposure and injury registration

All first team players in included teams were invited to participate in the study. Participation was voluntary and written informed consent was obtained at the time of study inclusion. At the beginning of every season, teams appointed a contact person within each respective medical team to be responsible for collecting data and communicating with the study group. During the study period, all individual player exposure during supervised training sessions and matches was recorded on standard attendance records. In addition, all time loss injuries that occurred were registered on standard injury cards containing information about type of injury and circumstances of the injury occasion (injury mechanism, affected side, time of injury, re-injury) (table 2).

Table 2 Definitions of variables used in the study

Training session	Team training that involved physical activity under the supervision of the coaching staff
Match	Competitive or friendly match against another team
Time loss injury	Any physical complaint sustained by a player that resulted from a football match or football training and led to the player being unable to take a full part in future football training or match play
Moderate injury	Injury causing 8–28 days absence
Severe injury	Injury causing >28 days absence
Re-injury	Injury of the same type and at the same site as an index injury
Injury incidence	Number of injuries per 1000 player-hours ((Σ injuries/Σexposure-hours)×1000)
Injury burden	Number of days absence per 1000 player-hours ((Σ days absence / Σ exposure-hours)×1000)

Each month, the appointed contact person reported the attendance records and injury cards to the study group. All injuries were given a diagnostic code by the study group in accordance with the Orchard Sports Injury Classification System (OSICS) 2.0.[18] OSICS 2.0 codes were used to identify isolated ankle syndesmotic injuries. Athletes with tenderness on palpation over the anterior interosseous membrane proximal to the ankle joint and positive special tests such as ankle external, rotation and syndesmosis squeeze test were suspected for syndesmotic injury. Uncertainty of the diagnosis was resolved through widening of the tibiofibular joint seen during radiographic assessment, or ultrasonographic or magnetic resonance imaging (MRI) evidence of rupture of syndesmotic ligaments without associated fibula fracture. Data collection was undertaken in accordance with a previously published consensus statement regarding how to conduct epidemiological research in professional football.[19] Methodology related to the exposure and injury registration has previously been described in detail.[19]

Data analysis and statistics

Data were analyzed using SPSS (IBM SPSS Statistics for Windows, V.23.0, IBM Corp, Armonk, New York, USA). Injury incidence was described as the number of injuries/1000 hours of exposure, with corresponding 95% confidence intervals (CI). Injury incidence in training and match play were calculated and rate ratio between training and match play were analyzed with Poisson regressions using match exposure hours as an offset. The proportion of match injuries occurring in different 15-minute- periods of match halves were compared to the expected 33% proportion, which would be present if injuries were evenly distributed between the different thirds, and analyzed with Z-statistics. Injury severity was defined by the number of days of absence caused by the injuries and described with mean (±SD) and median (25th and 75th percentile). Injury burden was defined as number of day's absence/1000 hours of exposure. Injury burden in training and match play were calculated and injury burden ratio between training and match play were analyzed with Poisson regressions using match exposure hours as an offset. The annual changes in injury incidence, injury burden and syndesmotic injury proportion (proportion of all ankle ligament injuries that were diagnosed as syndesmotic injuries) were analyzed using linear regression. In these analyzes injury incidence, injury burden and syndesmotic injury proportion were used as dependent variables in separate analyses, while season was used as the independent variable in all analyses. In addition, injury incidence, injury

burden and syndesmotic injury proportion in match play were also analyzed using linear regression with season included as the independent variable. Analyses of training injuries specifically were not performed since the number of injuries during training were few. To reduce possible effects of large temporary variations between seasons, moving averages (MA) of two consecutive seasons were also used as dependent variables in similar linear regression analyses. All analyses were two sided and the significance level was set at $p<0.05$.

RESULTS

Isolated syndesmotic injury incidence

The overall isolated syndesmotic injury incidence over the study period was 0.05 injuries per 1000 hours of exposure (95% CI 0.04 to 0.06) or 1 injury per team every 3 seasons. The injury incidence during match play was 13 times higher compared to the incidence during training, 0.21 (95% CI 0.16 to 0.26) and 0.02 (95% CI 0.01 to 0.02) respectively (RR 12.63; 95% CI 8.12 to 19.65). A significant annual increase in isolated syndesmotic injury incidence in general (R^2=0.495, b=0.003, 95% CI 0.001 to 0.004, p=0.003) as well as in match play (R^2=0.354, b=0.013, 95% CI 0.002 to 0.023, p=0.019) was observed over the 15 seasons (Figure 1).

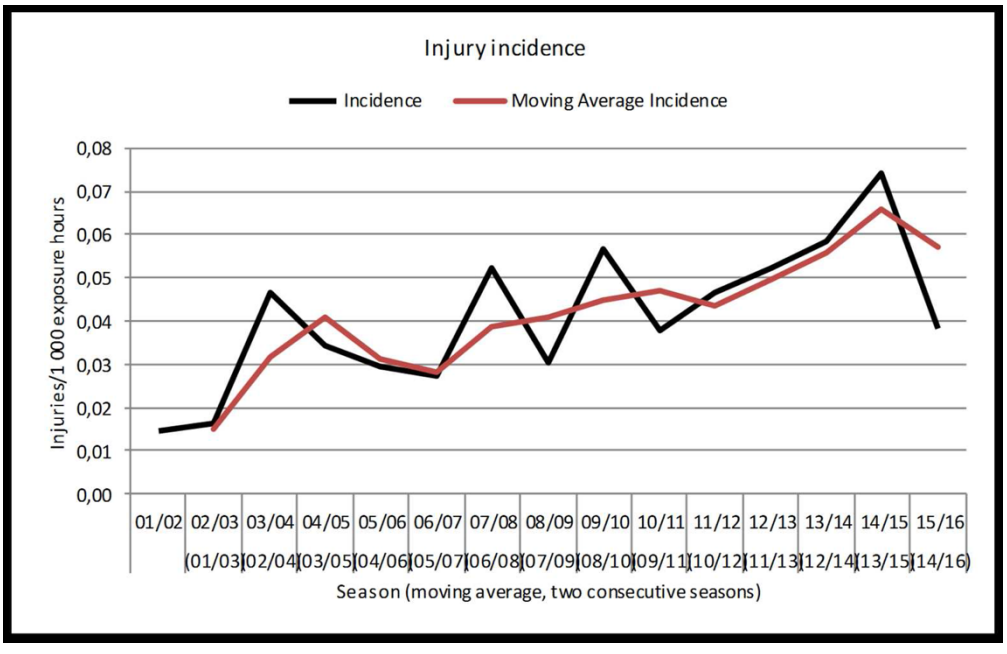

Figure 1: Seasonal variation in injury incidence of syndesmotic injuries in professional football. A significant 7.5% annual increase in isolated syndesmotic injury incidence was observed (R2=0.525, b=0.075, 95% CI 0.032 to 0.117, p=0.002) over the 15 seasons. MA, moving average;

The sensitivity analyses, using moving averages of two consecutive seasons, also showed an annual increase in syndesmotic injury incidence in general (R^2=0.822, b=0.003, 95% CI 0.002 to 0.004, p<0.001) as well as in match play (R^2=0.751, b=0.015, 95% CI 0.009 to 0.020, p<0.001).

Isolated syndesmotic injury proportion of all ankle ligament injuries

Out of the 14 653 injuries registered during the 15 seasons, 1950 (13%) affected the ankle with 1320 (9%) ankle ligament injuries.
Out of these 1320 injuries, 94 (7%) were diagnosed as syndesmotic injuries. An annual increase of the proportion of syndesmotic injuries (proportion of all ankle ligament injuries that were diagnosed as syndesmotic injuries) was observed (R^2=0.601, b=0.006, 95% CI 0.003 to 0.009, p=0.001) (Figure 2).

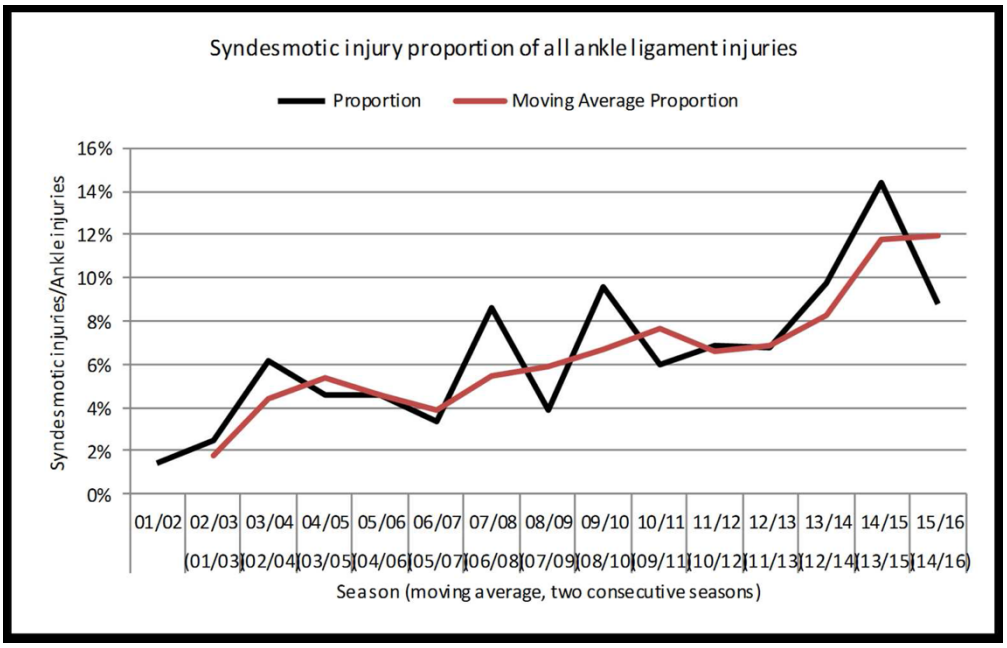

Figure 2: Seasonal variation of the proportion of syndesmotic injuries of all ankle ligament injuries in professional football. An annual 10.7% (R2=0.633, b=0.107, 95% CI 0.058 to 0.155, p<0.001) increase of the proportion of syndesmotic injuries was observed.
MA, moving average;

The proportion of syndesmotic injuries during match play also increased

annually (R^2=0.430, b=0.006, 95% CI 0.002 to 0.010, p=0.008).
The sensitivity analyses, using moving averages of two consecutive seasons, also showed an annual increase in the proportion of syndesmotic injuries in general (R^2=0.818, b=0.006, 95% CI 0.004 to 0.008, p<0.001) and in match play (R^2=0.758, b=0.006, 95% CI 0.004 to 0.009, p<0.001).

Injury patterns

Seventy percent of the syndesmotic injuries occurred during match play and the remaining 30% during training. Being tackled was responsible for one third of the syndesmotic injuries. The remaining injuries were accounted for by: twisting/turning (13%), landing from a jump (10%), collisions (5%), being kicked (5%), tackling (4%), other (10%), and for 20% the mechanism was unknown. Seventy-four percent of the injuries involved contact of some kind and 54% affected the dominant leg (defined as the preferred kicking leg). Seven percent were considered re-injuries. No significant differences were found between the proportion of injuries occurring during 15-minute-periods of each half (0-15, 16-30, 31-45 minutes) and the 33% which would be expected if the injuries were evenly distributed between the different thirds of the match halves (Figure 3).

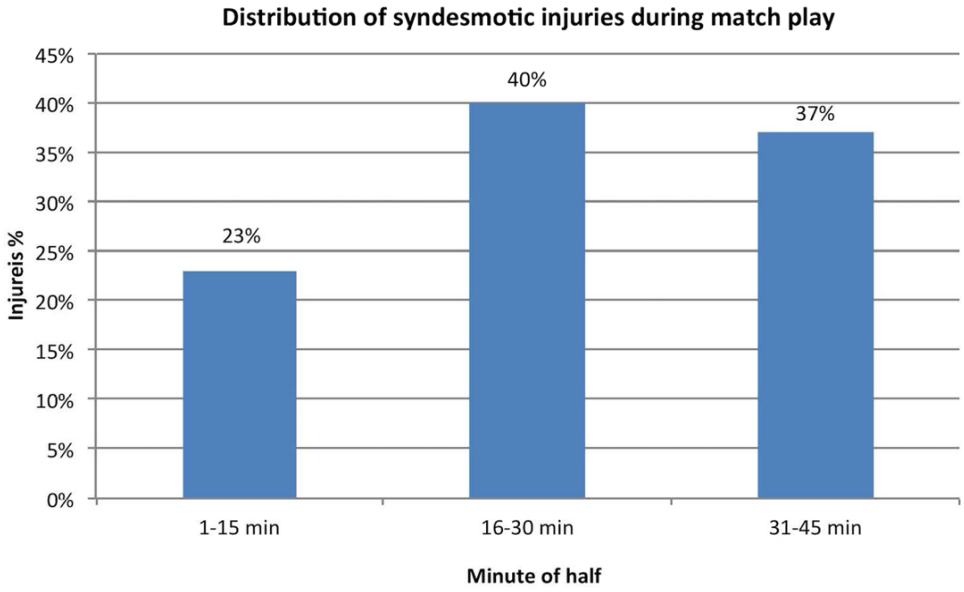

Figure 3: Distribution of syndesmotic injuries during 15 min periods of match play in professional football;

158x96mm (300 x 300 DPI)

Injury severity and absence

More than 90% of the syndesmotic injuries were classified as moderate to severe (causing more than 1 week absence) with 57% being severe (causing more than 1 month absence). The mean (SD) absence following a syndesmotic injury was 39 (28) days and the median (25th, 75th percentiles) was 34 days (19, 52).

Isolated syndesmotic injury burden

A total of 3,652 days of absence due to syndesmotic injuries were reported over the study period, representing an injury burden of 1.8 days absent per /1000 hours of exposure.

The injury burden due to match exposure was 18 (RR 18.22; 95% CI 16.86 to 19.68) times higher compared to training (8.8 days absent/1000 match hours versus 0.5 days absent per 1000 training hours).

There were no significant annual changes in injury burden in general (R^2=0.033, b=0.032, 95% CI -0.073 to 0.138, p=0.520) or in match play (R^2=0.003, b=0.060, 95% CI -0.598 to 0.718, p=0.847).

Similarly, no annual change in general (R^2=0.059, b=0.028, 95% CI -0.043 to 0.099, p=0.405) or in match play (R^2=0.005, b=0.050, 95% CI -0.389 to 0.488, p=0.809) was shown when the two-season moving average of injury burden was analyzed (Figure 4).

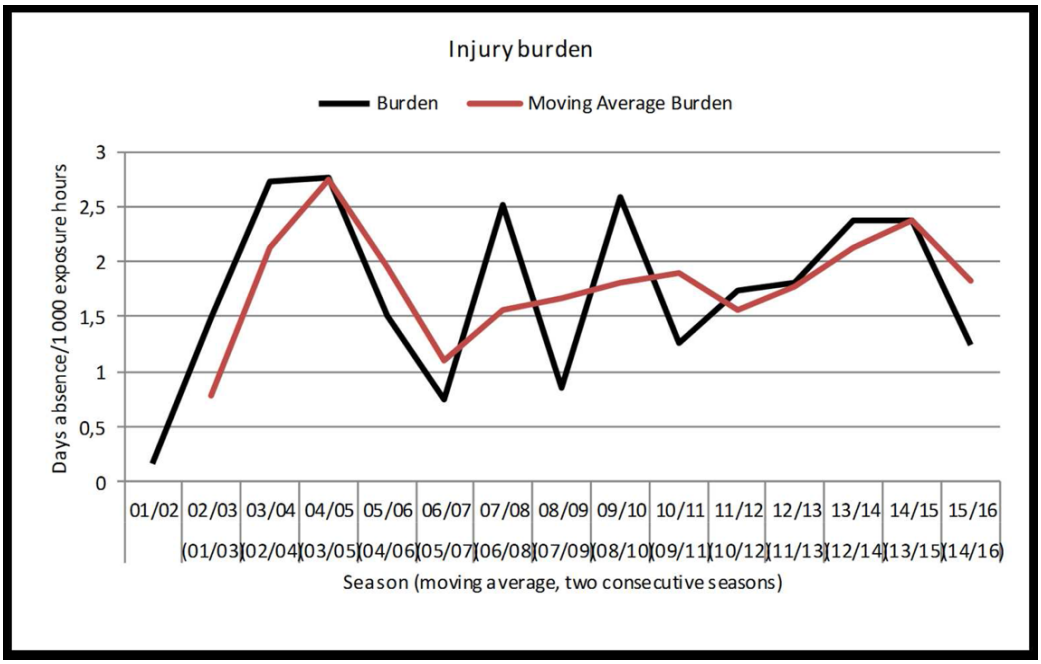

Figure 4: Seasonal variation in injury burden of syndesmotic injuries in professional football. There were no significant annual changes in injury burden (R2=0.123, b=0.058, 95% CI -0.035 to 0.151, p=0.200). Similarly, no annual change was shown when the two season moving average of injury burden was analyzed. MA, moving average;

DISCUSSION

The incidence figures indicate that an isolated syndesmotic injury in professional football is a relatively rare event. Despite this, the injury incidence during match play seems to have increased over the past 15 seasons. Return to play after injury took on average greater than 5 weeks.

Comparison with Other Sports

Isolated syndesmotic injuries are more common in collision sports and those that involve rigid immobilization of the ankle in a boot.[20] In a cohort consisting of National Collegiate Athletic Association (NCAA) American football players the incidence of syndesmotic injury during games was 1.6 per 1000 athlete exposures (defined as one athlete participating in one practice or competition in which there was a possibility for athletic injury).[21] Flik et al.[8] collected injury data from 12 NCAA Division I ice hockey teams over one season and found that the game injury was 0.93 per 1000 athlete exposures. For rugby the injury rate per 1000 hours of exposure was 0.89 in Rugby Union and 0.46 in Rugby

League.[22] In our study, the syndesmotic injury incidence rate during match play over the study period was 0.21 injuries per 1000 hours of exposure. Accounting 90 minutes exposure for each match played, the incidence of syndesmotic injury during games was 0.32 per 1000 athlete exposures. Hence, the risk of incurring a syndesmotic injury playing football is lower compared to American football, ice hockey or rugby.

Professional versus non-professional football players

Mauntel et al.[15] described the epidemiology of isolated syndesmotic injuries among college student-athletes in 25 sports over six seasons. Similar with our findings, the injury incidence during match play was 0.34 per 1000 athlete exposures and the injury incidence during training was 0.047 per 1000 athlete exposures. Compared to professional players, fewer injuries were caused by contact (74% among professional players versus 56% among non-professional players). Interestingly, the absence following an isolated syndesmotic injury was in 80% of the injuries less than 21 days. Previous studies, including ours have described an absence ranging from 30 to 62 days.[23-26] A possible explanation could be that some lateral ankle sprains may have been misdiagnosed and diagnosed as syndesmotic injuries instead. Unfortunately, the authors did not describe the methods used for diagnosing the injury.

Yearly Increase in Injury Incidence

Over the 15 seasons an annual increase in isolated syndesmotic injury incidence in general (0.003 injuries per 1.000 hours) as well as in match play (0.013 injuries per 1.000 hours) was observed (Figure 1). This might be a reflection of the fact that today's health care providers have greater suspicion for the injury, or perhaps because of the more frequent use of magnetic resonance imaging or diagnostic arthroscopy.[24,27] Another explanation could be a general philosophical change in the way clubs attend to player complaints.[28] Nonetheless, our data showed that being tackled caused most injuries, and the injury happened 13 times more frequently during match play then during a training session. Hence, we propose that the increase in annual injury incidence is caused by a more aggressive style of play during matches over the 15 years.

Low proportion of syndesmotic injury

Isolated syndesmotic injuries accounted for 7% of all ankle ligament injuries. The reported proportion of isolated syndesmotic injuries among overall ankle ligament injuries ranges from 18 to 74%.[8,10,21,25,29-31] This variation can be explained by the fact that some sports have extrinsic risk factors associated with syndesmotic injury. Skiers and ice hockey players wear boots causing rigid immobilization of the ankle leading to high-torque external rotation of the foot,[5,8,25] and American football is often played on artificial turf instead of natural surfaces.[10,21,29,31] Another plausible explanation is that an isolated syndesmotic injury can be frequently misdiagnosed as an ankle sprain.

Injury burden

The average absence from play following a syndesmotic injury was 39 days. This is in line with findings from previous studies that reported prolonged time to return to play after a syndesmotic injury, ranging from 30 to 62 days.[23-26] In contrast, following lateral ankle sprains the absence has been reported 15 days.[32] In addition, over the course of 15 seasons we found no change in injury burden despite the injury incidence having increased. Hence, the time to return to play after injury over the past 15 seasons has decreased. A reason for this decrease could be that recent research on treatment strategies and diagnosing the severity of the injury may have led to improved outcomes.[11,23,33-35] Nevertheless, to reduce the risk and consequences of this injury to a team, club medical staff should be conscious of whether the injury is stable or unstable since each requires different treatment strategies.[11] Appropriate management of syndesmotic injuries leads to an earlier return to play.[23] In addition, use of ankle braces,[36] referees being stricter while judging player tackling, and changes to game play rules -such as sliding- may help reducing the injury incidence rate.

Strengths and study limitations

The strength of this study is the large homogenous data set prospectively collected among 61 professional football teams. Having many teams working together provides robust data from which to draw conclusions.[16,37] There are, however, a few limitations.
We were not able to capture data on possible confounders and these could therefore not be included in our analyses. First, the injury form did not capture the examination findings or diagnostic tests results to classify syndesmotic

injuries beyond identifying a lack of fracture, or provide information on associated injuries. Second, the diagnosis was made by the medical staff of each football team and thus subject to the biases and experience of different physicians. Increasing awareness of the diagnosis of 'syndesmosis injury' may explain part of the trend to increased incidence. Third, we did not capture data on pitch or weather conditions at the time of injury. Fourth, we did not capture data on player medical history (i.e., previous syndesmotic or ankle ligament injury). Fifth, there was no information available on how players were treated (i.e., conservatively or surgically). These data would have been useful to provide better perspective with respect to interpreting absentee time following the injury.

CONCLUSION

Our findings indicate a significant increase in the incidence of isolated syndesmotic injuries in professional football players. We speculate this is likely caused by more aggressive playing style during matches. The average return to play time following injury exceeded 5 weeks, and there was no change found in injury burden over 15 seasons. We recommend club medical staff to be conscious of the nature of the injury to reduce the consequences of such injuries to a team.

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CHAPTER

2

THERE'S NO SUCH THING AS A SIMPLE ANKLE SPRAIN

CHAPTER 2:

There's no such thing as a simple ankle sprain

Ligamentous Injuries and the Risk of Associated Tissue Damage in Acute Ankle Sprains in Athletes: A Cross-sectional MRI Study.

American Journal of Sports Medicine (2014)

Roemer F, Jomaah N, Niu J, Almusa E, Roger B, D'Hooghe P, Geertsema C, Tol J, Khan K, Guermazi A

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Epub 2014 Apr 21

ABSTRACT

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.

INTRODUCTION

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 (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.3[4] 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 3 224–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 3 224–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 3 224–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 3 224–pixel matrix; 1 NEX; 15.9-cm2 FOV; 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 3 224–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 intra-observer 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.
Intra-observer 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 = peri-ligamentous 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 peri-ligamentous edema).[18,22] The ATFL, CFL, and PTFL were assessed separately.
- The syndesmotic ligaments were assessed in 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 tibio-spring ligaments (the latter scored together with the superficial deltoid) (0-4 scale, 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 infero-plantar longitudinal, medio-plantar oblique, and supero-medial)
- 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; 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).

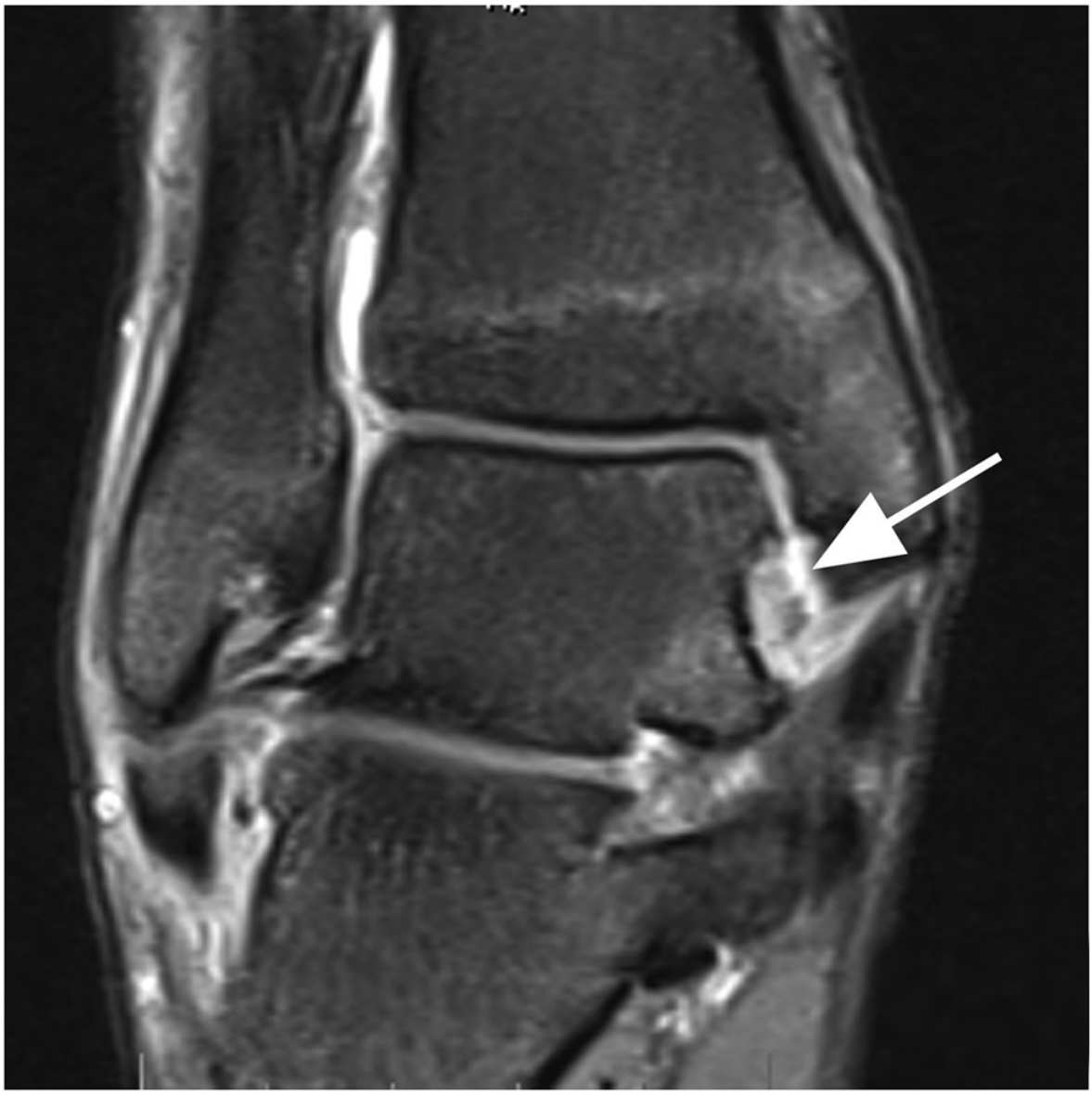

Figure 1. Deltoid ligament injury. Coronal proton density– weighted turbo spin echo magnetic resonance imaging shows complete disruption (grade 3) of the posterior tibiotalar ligament (arrow).

- 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 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 >50% of maximum capsular distension (Figure 4).

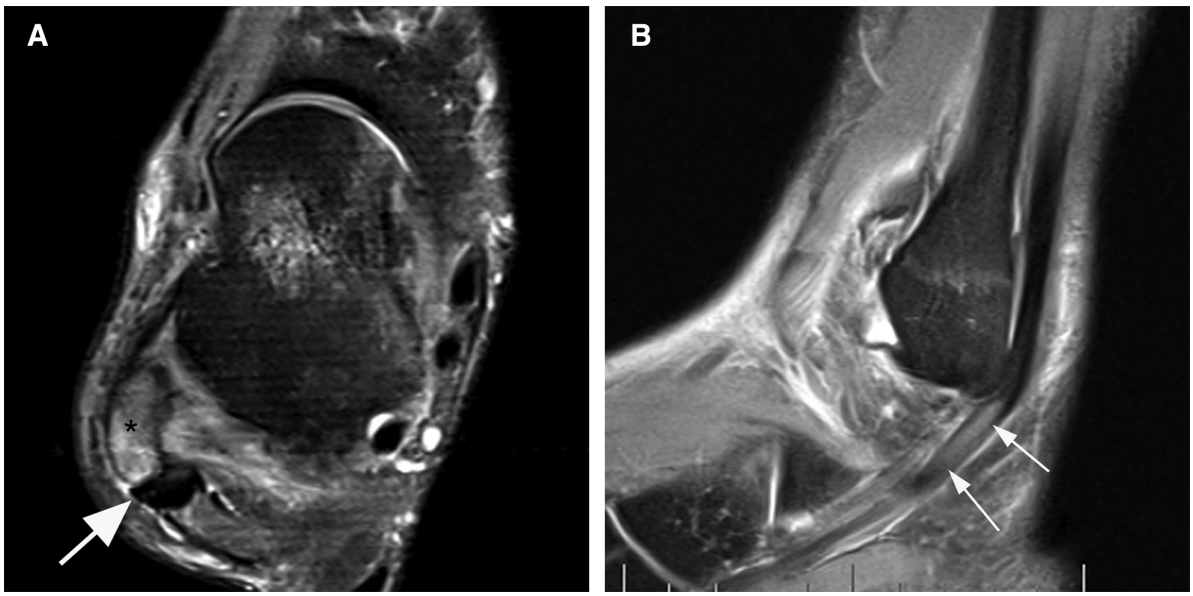

Figure 2. Tendon and retinaculum damage. (A) Axial proton density–weighted turbo spin echo magnetic resonance imaging (MRI) shows the beginning of subluxation of the peroneus brevis tendon laterally due to a grade 1 retinaculum injury with stripping of the superior peroneal retinaculum off the distal 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.

Analytic Approach

Six exclusive, different injury patterns were defined based on lateral and syndesmotic ligament involvement:
(1) no lateral ligament injury, no syndesmosis injury (non-injured 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
(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 syndesmotic damage (patterns 1 and 2)
(2) ankles with complete ATFL injuries but no syndesmotic involvement (patterns 3 and 4)
(3) ankles with partial or complete syndesmotic disruption (patterns 5 and 6).

Ankles with no or only low-grade lateral ligament injuries and no syndesmotic damage were used as the reference group. Results were adjusted for age and sex. Reliability was assessed using weighted k 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 6 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 6 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 syndesmotic damage, 103 (39.5%) had complete ATFL injuries (and any CFL or PTFL grade) and no syndesmotic injuries,

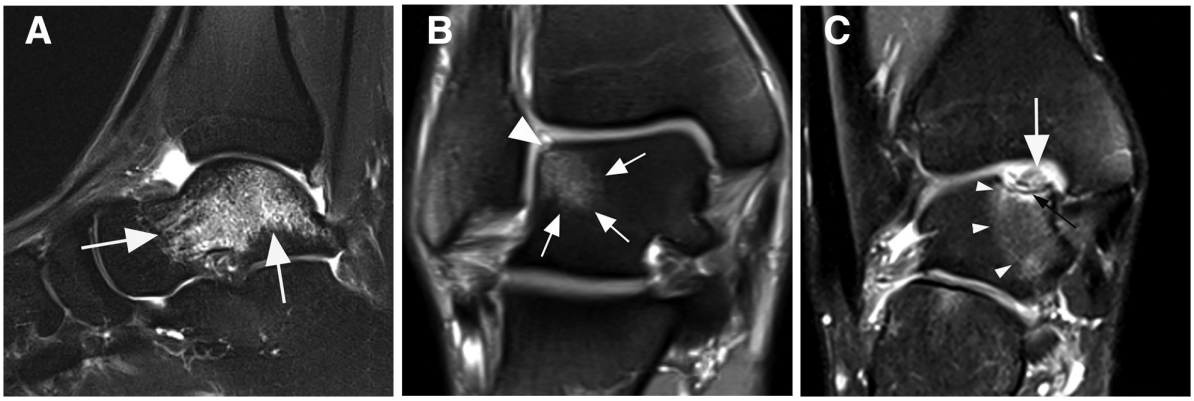

Figure 3. Bone injury on proton density–weighted magnetic resonance imaging. (A) A large talar bone contusion (grade 2) involving the talar body and talar neck is shown (arrows). No fracture line is seen. (B) Acute osteochondral injury to the lateral talar shoulder (grade 4). In addition to the bone 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).

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 detailed in Table 1.

There were no statistical differences in frequencies of the injury patterns for age or sex. Intra-observer 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 intra-observer 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).

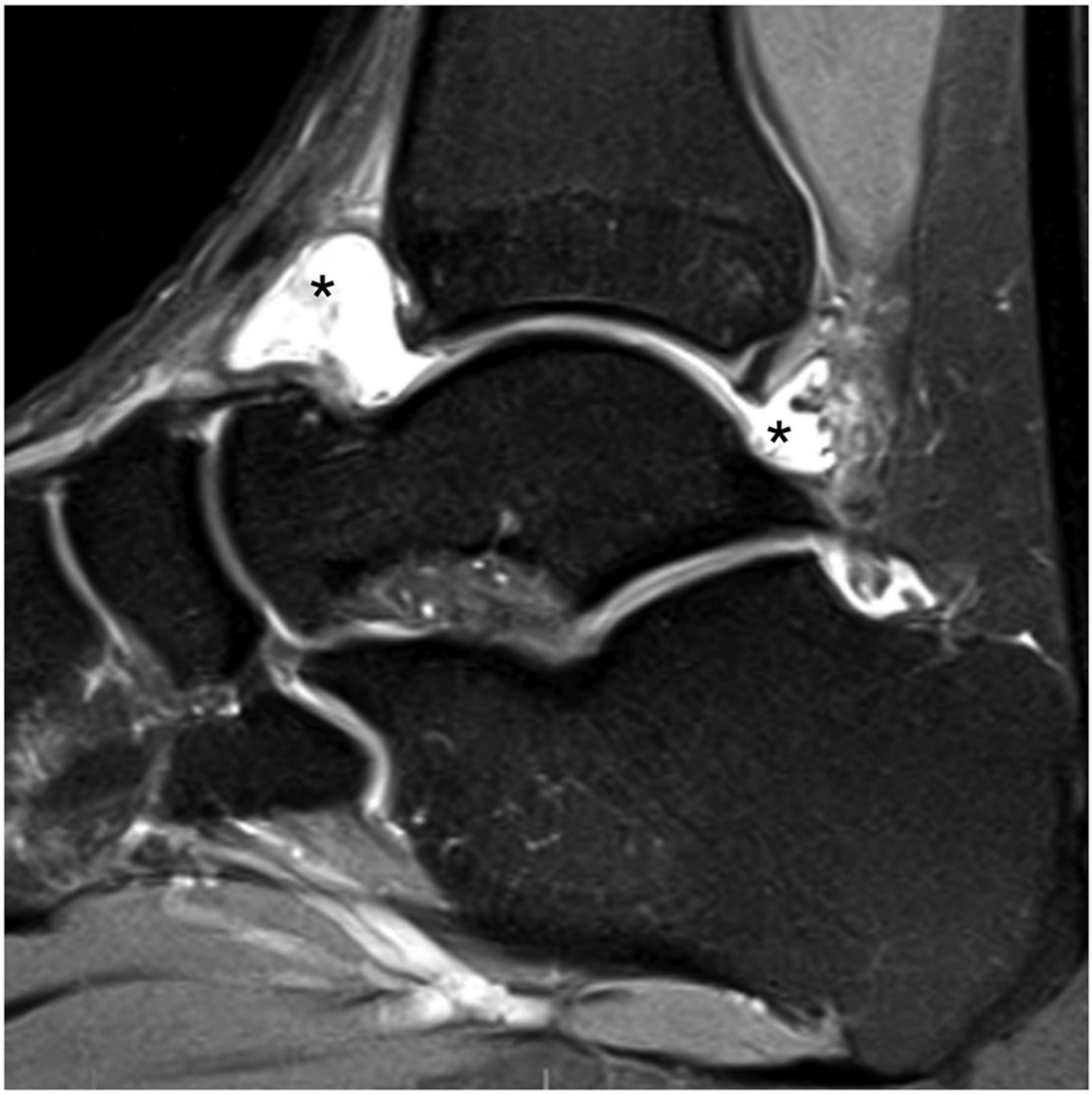

Figure 4. Traumatic joint effusion. Sagittal proton density–weighted fat-saturated magnetic resonance imaging shows a large amount of joint effusion anteriorly and posteriorly at the tibiotalar joint (asterisks).

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)
P value (Fisher exact test)		.81				.76			

[a]Values are expressed as n (%). ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; PTFL, posterior talofibular ligament.
[b]All 3 lateral ligaments = 0, 1, or 4; all 4 syndesmotic ligaments = 0, 1, or 4. Grade 4 (scar) is treated as normal (equal to 0 or 1), assuming functional stability of scar lesions.
[c]ATFL = 1 or 2 and/or CFL = 1 or 2; and PTFL = 0, 1, 2, or 4; all 4 syndesmotic ligaments = 0, 1, or 4.
[d]ATFL = 3; CFL = 0, 1, or 4; PTFL = 0, 1, or 4; all 4 syndesmotic ligaments = 0, 1, or 4.
[e]ATFL = 3; CFL = 2 or 3; PTFL = 0, 1, 2, 3, or 4; all 4 syndesmotic ligaments = 0, 1, or 4.
[f]ATFL = 2 or 3; CFL = 2 or 3; PTFL = 0, 1, 2, 3, or 4; at least 1 of 4 syndesmotic ligaments = 2 or 3.
[g]ATFL = 0, 1, or 4; CFL = 0, 1, or 4; PTFL = 0, 1, or 4; at least 1 of 4 syndesmotic 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

[a]Anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament scored from 0-4.
[b]Anterior-inferior tibiofibular, posterior-inferior tibiofibular, interosseous membrane, and transverse ligament scored from 0-4.
[c]Superficial/tibiospring and deep bundles of the deltoid ligament scored from 0-4.
[d]Inferoplantar longitudinal, medioplantar oblique, and superomedial scored from 0-4.
[e]Interosseous calcaneal and cervical ligaments.
[f]Peroneal, flexor, and extensor retinacula.
[g]Fibula, tibia, calcaneus, navicular, and other scored from 0-2; talar osseous lesions scored from 0-5.
[h]Peroneal, flexor, and extensor tendons scored from 0-3.
[i]Tibiotalar and talocalcaneal joints scored from 0-2.

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

Injury Pattern[c]	No. of Ankles	Associated Injuries: Ligaments/Tendons[b] 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)

[a]Values are expressed as n (%). ATFL, anterior talofibular ligament; CFL, calcaneofibular ligament; PTFL, posterior talofibular ligament.
[b]Any lesion ≥1.
[c]See Table 1.

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).

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 syndesmotic 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

[a]Values 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.
[b]Any lesion ≥1.
[c]Rows 1 and 2 combined from Table 3.
[d]Adjusted for age and sex.
[e]Rows 3 and 4 combined from Table 3.
[f]Rows 5 and 6 combined from Table 3.

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). Additional osseous involvement ranged from 3.4% (navicular) to 23.0% (tibia) with no fractures but contusions only. There was an increased risk for tibial osseous involvement for ankles with any syndesmotic injury (aOR, 4.46; 95% CI, 1.79-11.11). 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 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). The risk for talar bone contusions increased for ankles with any syndesmotic injury but not for ankles with lateral ligament injuries (aOR, 3.43; 95% CI, 1.72- 6.85).

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 syndesmotic involvement in 56% of patients and syndesmotic injuries with or without lateral ligament damage in about 20%. The most common findings with regard to associated 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 syndesmotic injury but not for ankles with lateral ligament injuries. The risk for small talar bone contusions increased for ankles with any syndesmotic 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 colleagues[27] 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%.[25]

Takao et al, 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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From “Low” to “High” Athletic Ankle Sprains: A comprehensive Review.

Operative Techniques in Orthopaedics Journal (2018)

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From “Low” to “High” Athletic Ankle Sprains: A Comprehensive Review
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Operative techniques in orthopaedics, 2018
https://doi.org/10.1053/j.oto.2018.01.002

ABSTRACT

Generally, most Grade I-III acute lateral ligament injuries can be treated conservatively. Yet despite a propensity of research regarding ankle sprains some controversy still exists as regarding the optimum treatment of grade III injuries in athletes.

Physical exercise therapy combined with progressive weight bearing is a fundamental component of the functional treatment of acute lateral ligamentous injury.

Generally, early active range of motion exercises is followed by strengthening exercises, proprioception, and functional exercises. Most re-injuries are probably related to inadequate neuromuscular training during the rehabilitation phase.

Treatment of grade III lateral ligament injury especially in athletes remains controversial. Reviews comparing surgery vs conservative treatment have failed to demonstrate a clearly superior method. Thus, functional treatment might be preferred over surgery in most cases. However, surgical treatment may be beneficial in certain professional athletes on an individual basis.

The advantage of surgical repair is significantly less objective when compared to non-operative treatment and this factor has been found to be predictive for future ankle sprains.

Recent arthroscopic surgical techniques have been described as part of the therapeutical options in the treatment of mainly chronic ankle instability. Also, new data on the role of the calcaneo-fibular ligament in this regard highlights key points that need to be addressed before deciding for optimal treatment.

INTRODUCTION

Low ankle sprains have an estimated of 30,000 per day in the USA[1] that accounts for almost 2 million per year and similar numbers appear for Europe.[2] In addition, 20%-40%of all sports-related injuries in the USA are ankle sprains.[3] This high incidence of ankle sprains can be partly explained by the natural tendency of the ankle joint to go into inversion, and the relative weakness of the lateral ligaments. The most common mechanism of injury is inversion of a plantar-flexed foot. An ankle sprain can be defined as any tear to the ankle ligaments and can range from microscopic, to complete tears.[4]

Syndesmotic ligament injury is a special subset of ankle sprains, and often is referred to as a "high ankle sprain." In comparison therefore, we might use the term "low" ankle sprain, sprains while referring to lateral ankle ligament sprains.

The anterior talo-fibular ligament (ATFL) is the most commonly injured ankle ligament during a "low" ankle sprain, accounting for almost 90%-95%.[5] With more severe injury progression, rupture of the ATFL is followed by injury to the calcaneo-fibular ligament (CFL) and lastly, generally in case of a serious trauma, to the posterior talo-fibular ligament (PTFL). Recently, a magnetic resonance imaging (MRI) study demonstrated that 41% of the patients with an ankle inversion injury, damaged both the ATFL and CFL, whereas only 5% had injured the PTFL.[6]

Return to activity after a sustained ankle sprain has been shown to be dependent on the severity of the initial injury and the presence of any concomitant pathology.[7] High rates of re- injury after a primary sprain have been shown, with up to 34% of patients suffering a second sprain within 3 years of their initial injury.[7]
Repeated ankle sprains can lead to attenuation of the ATFL and the overall lateral ligamentous complex. This may render those tissues incompetent and leads to chronic ankle instability that can supervene in 10%-20% of the cases.[8] Up to 40% of the patients in the general population will report residual symptoms after classic treatment for an acute ankle sprain[7,9]; including chronic pain and recurrent instability. "High" ankle sprains are reported to occur in 1%-18% of patients with an ankle sprain.[5,10] However, this is probably an underestimate, as 20% of athletes with an acute ankle sprain have evidence of syndesmotic injury on MRI.[11] Male gender, elite performance, and a plano-valgus alignment are risk factors for syndesmotic injury in athletes.[12,13]
Syndesmotic injuries can occur with ankle sprains only, fractures, or both. In fact, 23% of ankle fractures are reported to have combined syndesmotic injuries.[14] The associated fractures are commonly either of the fibula or of the posterior and medial malleoli. Syndesmotic injury should be increasingly suspected if there is an associated fracture of the proximal fibula (Maisonneuve fracture, Figure 1) and they are associated with prolonged pain, disability, and an unpredictable time away from sports.[15]

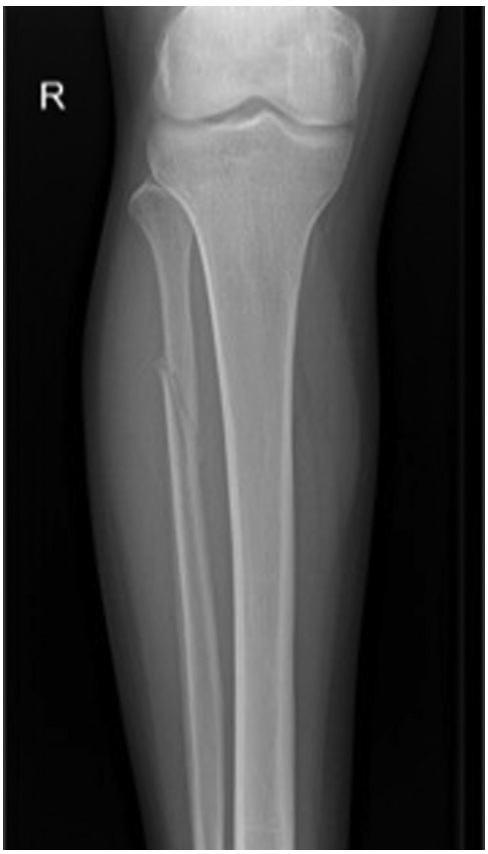

Figure 1. Maisonneuve fracture.

The general mechanism of injury for syndesmotic ankle sprains is a forceful external rotation of the foot and ankle with the ankle in dorsiflexion and the foot pronated.[16] Whilst the talus rotates in the mortise, the fibula rotates externally, moves posteriorly and laterally, separating the distal tibia and fibula. This will sequentially cause tears of the anterior inferior tibio- fibular ligament (AITFL), the deep deltoid ligament or might alternatively cause a malleolar fracture. This shall be in turn followed by a tear of the interosseous ligament (IOL) and finally the posterior inferior tibio-fibular ligament (PITFL).[16,17]

Severity of syndesmotic injury varies, ranging from a partially torn AITFL to a complete disruption of all ligaments with mortise widening. It has been shown that combined deltoid and syndesmosis injury will critically compromise talar stability.[18] The magnitude of force and its duration will determine the extension of syndesmotic and interosseous injury proximally[13] and this may eventually lead to a Maisonneuve fracture. Another injury mechanism for syndesmotic ankle sprains is hyper-dorsiflexion. Forced dorsiflexion of the ankle causes the wider anterior talus to act as a wedge that can cause injury to the syndesmotic ligaments.

CLINICAL FEATURES

"Low" Ankle Sprain

Clinically, patients will recount a sudden twisting of the ankle. Those with lateral ligamentous rupture report more immediate swelling and are more frequently obliged to halt their activities, compared to those without a rupture.[19] Ankle sprains usually are accompanied by an audible snap or crack. In a recent systematic review, it was found that application of the Ottawa rules is highly valuable for excluding coexisting fractures.[20] ATFL laxity could be evaluated by the anterior drawer test, whereas the talar tilt test helps in recognizing CFL instability. However, manual stress tests might be less reliable in the acute phase, because of pain and swelling. A delayed physical examination (4-5 days) has been shown to give better diagnostic results and is considered the gold standard in the diagnosis of acute lateral ligament injury, with a sensitivity of 96% and a specificity of 84%.[21,22]
On the other hand, the presence of "high ankle pain and tenderness," more proximally, is suggestive of a more significant injury.[23] In fact, it has been shown that there is a significant correlation between how far this tenderness radiates proximally in the leg and the severity of the injury and consequently, the time to return to sports.[23] Patients with high ankle sprains, may complain of the inability to bear weight, swelling, pain during the push off phase of gait and pain anteriorly between distal tibia and fibula, as well as postero-medially at the level of the ankle joint.[15] Ankle ROM will often be limited, with pain felt more at terminal dorsi- flexion.[24] Numerous special tests are used to detect syndesmotic injury. However, a recent systematic review on 8 different tests reported a low diagnostic accuracy of these tests.[25] The squeeze test was the only test with a clinical significance.[25]

In the diagnosis of ankle sprains, the Ottawa ankle rules are very useful to rule out fractures, with a sensitivity of almost 100%.[26] Conversely, stress radiographs are usually not suggested for the routine diagnosis of lateral ligament injury, as they are difficult to perform and will not alter the management. Both ultrasonography and MRI can be valuable in diagnosing any concomitant chondral or tendon injury. Recently a study compared ultrasonography in the emergency room with MR images for injuries of the ATFL and found no differences in diagnostic accuracy.[27] The sensitivity and specificity of MRI in diagnosing ATFL injuries are 92%-100% and 100%, respectively.[28,29]

High ankle sprain

In the diagnosis of syndesmotic injuries—if there is a clinical suspicion of a Maisonneuve fracture (Figure 1) —full length radiographs of the lower leg are indicated. Several radiographic parameters have been developed to help identify syndesmotic injuries: the tibiofibular clear space which represents the distance between the medial border of the fibula and the lateral border of the posterior tibia, providing the most reliable indicator of a syndesmotic injury.[30]

Computed tomography (CT) is useful in detecting small avulsion fractures and is considerably more accurate than radiographs in revealing subtle diastasis.[31] Recently, bilateral standing CT is developing as an alternative diagnostic stress view, although prospective comparatively controlled data is still currently lacking.[32]

MRI has been considered the investigation of choice for suspected syndesmotic ligament injury.[33] It demonstrated a sensitivity of 100% and a specificity of 93% for AITFL injuries and sensitivity and specificity of 100% for PITFL tears.[34] In a retrospective MRI study, a high prevalence of associated injuries was found, comprising osteochondral lesions (28%), bone contusions (24%), and osteoarthritis (10%).[35] There are still no reports that have correlated the extent of these lesions on imaging and the recovery time or clinical outcome. Although dynamic ultrasonographic examination showed a 100% sensitivity and specificity[36], unfortunately it has the drawback that it lacks the ability to detect associated injuries and is investigator dependent.[33]

THERAPEUTICAL OPTIONS

"Low" Ankle Sprain

The definitive management of ankle sprains shall depend to a large extent upon the classification of the injury.[37] In "low" ankle sprains this classification combines actual ligament damage with patient's symptoms and is of more significance with a delayed physical examination. Grade I (mild) injuries are a stretch of the ligament without macroscopic rupture. There is minimal swelling and tenderness, and no increased laxity. Grade II (moderate) injuries include partial tear of the ligaments, with moderate pain, swelling and tenderness. There is a mild to moderate increase in laxity, some loss of motion, and moderate functional disability. In grade III (severe) injuries (Figure 2A and B), a

complete rupture of the ligaments is present with severe pain, swelling, and bruising.
There is increased laxity and a major loss of function. The patient is also usually unable to bear weight.
Generally, most Grade I-III acute lateral ligament injuries can be treated conservatively. Yet despite a propensity of research regarding ankle sprains some controversy still exists as regarding the optimum treatment of grade III injuries in athletes.[38]
The initial treatment of lateral ankle ligament sprains usually involves the RICE-principle (rest, ice (cryotherapy), compression, and elevation), for the first 4-5 days; although a recent systematic review found no conclusive value for the application of that principle.[39]
Manual mobilization of the ankle was found to add limited value and therefore is discouraged. Additionally, no benefit was found for the usage of laser therapy, ultrasound therapy, or electrotherapy.[40] Functional treatment was proven to be more beneficial than long periods of immobilization and the use of NSAIDS, taping or orthosis is valuable in the initial phase.[38,41] However, for severe (Grade III) lateral ligamentous injuries, a short period of immobilization (max 10 days) in a below knee cast or a removable boot could be advantageous.[38,42]
Controlled stresses on an injured ligament promotes more proper collagen fiber orientation, and consequently, the use of an external ankle support is encouraged. To this effect, a recent study found no differences in outcome between tape, semi-rigid brace and a lace-up brace 6 months after treatment[43], however, most studies report superior results for protection with a brace.[38,44]
Physical exercise therapy combined with progressive weight bearing is a fundamental component of the functional treatment of acute lateral ligamentous injury.[45]
Rehabilitation programs for acute lateral ligamentous injuries, based on current best evidence, have been described.[46–48] Generally, early active range of motion (ROM) exercises is followed by strengthening exercises, proprioception, and functional exercises. Most re-injuries are probably related to inadequate neuromuscular training during the rehabilitation phase.[45]

Treatment of Grade III lateral ligament injury especially in athletes remains controversial. Reviews comparing surgery vs conservative treatment have failed to demonstrate a clearly superior method.[38,44] Thus, functional treatment might be preferred over surgery in most cases.[38,44] However, surgical treatment may be beneficial in certain professional athletes on an individual basis.[49] The advantage of surgical repair is significantly less objective instability when

compared to non-operative treatment[45] and this factor has been found to be predictive for future ankle sprains.[50] A recently described rehabilitation regimen for lateral ligament injuries after direct anatomic reconstruction included 1 or 2 weeks in below knee cast, then 2-4 weeks in a walking boot. This was then followed by an active rehabilitation protocol with the use of an ankle support.[51]

"High" Ankle Sprain

The classification of syndesmotic injury is divided into 3 Grades: Grade I is a minor sprain to the AITFL without instability; Grade II represents a tear of the AITFL and a

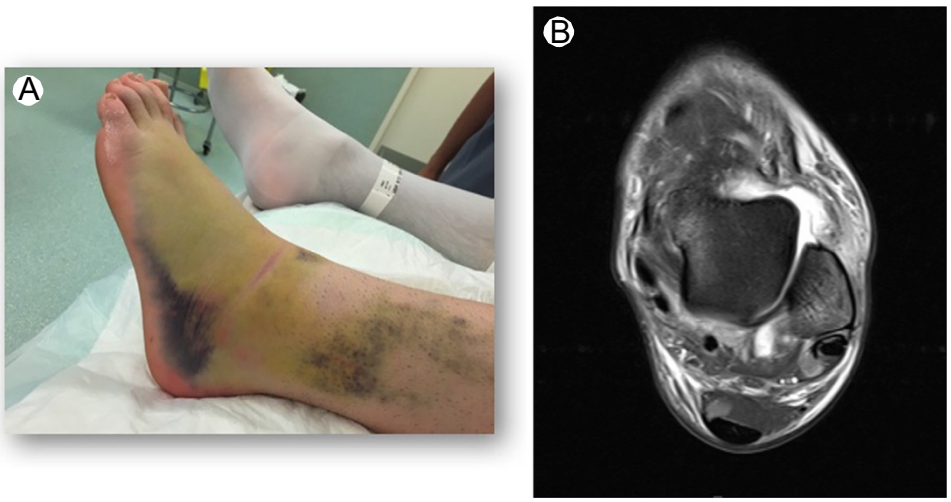

Figure 2. (A) Clinical presentation of a grade 3 "low" ankle sprain. (B) Axial T2 MRI image of a grade 3 "low" ankle sprain.

Partial tear of the IOL with some instability; Grade III involves complete rupture of all syndesmotic ligaments.[33] Grade I injuries are usually treated with non-surgically.[52]

A 3-phase approach has been advocated[23,53]: an acute phase, a subacute phase, and an advanced training phase, delivered over a period of 2-3 weeks.

Treatment of Grade II injuries depends on syndesmotic stability.[33] A recent study in athletes with a stable syndesmosis, found that a positive squeeze test and injury to the ATFL and CFL are important factors in differentiating stable (type IIa) from dynamically unstable Grade II injuries (type IIb).[54] Recreational individuals without diastasis can be treated non-operatively with good results.[55] Compared to a lateral ankle sprain, the recovery time of a conservatively treated Grade IIa syndesmotic injury is more prolonged. In higher level

professional athletes, with a Grade II injury and clinical or radiological suspicion of dynamic instability (type IIb) an examination under anesthesia and arthroscopic visualization of the syndesmosis is recommended.[55,56] Dynamic diastasis of 2 mm or more merits fixation.[52] The conservative treatment for "high" ankle sprains consists of similar rehabilitation strategies as the "low" ankle sprains (proprioception, stability, taping/orthosis, and NSAIDS) like with the exception that no preventative strategies are available and that the time to return to play is over 5 weeks minimum.

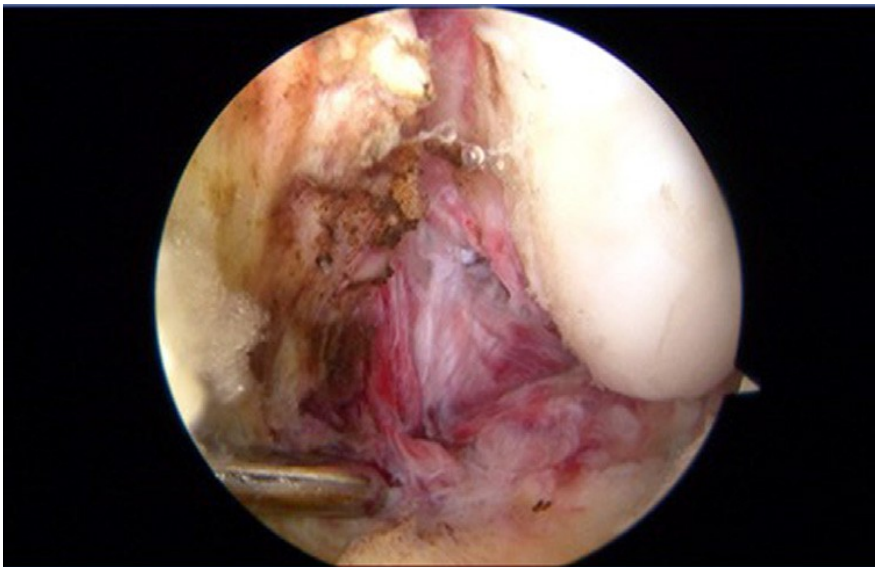

Figure 3. Arthroscopic view of a grade 3 syndesmotic injury.

Grade III injuries (Figure 3) will generally require operative fixation to maintain anatomic reduction of the ankle mortise. Screws or suture-buttons can both be used to stabilize the syndesmosis, with similar outcomes; but suture-button devices might provide the added value of a quicker return to play and a lower rate of implant removal.[57,58]

Arthroscopic visualization can identify and address any additional intra-articular pathology. Furthermore, it can be used to confirm anatomic reduction of the syndesmosis.[34]

Recent literature indicates that the routine removal of the screw is no longer advocated.[58] Syndesmotic ruptures are commonly associated with ankle fractures. After reduction and fixation of the associated fracture, intraoperative testing of syndesmotic stability should be performed. The Hook or Cotton test are considered as the most reliable intraoperative stress tests.[59]

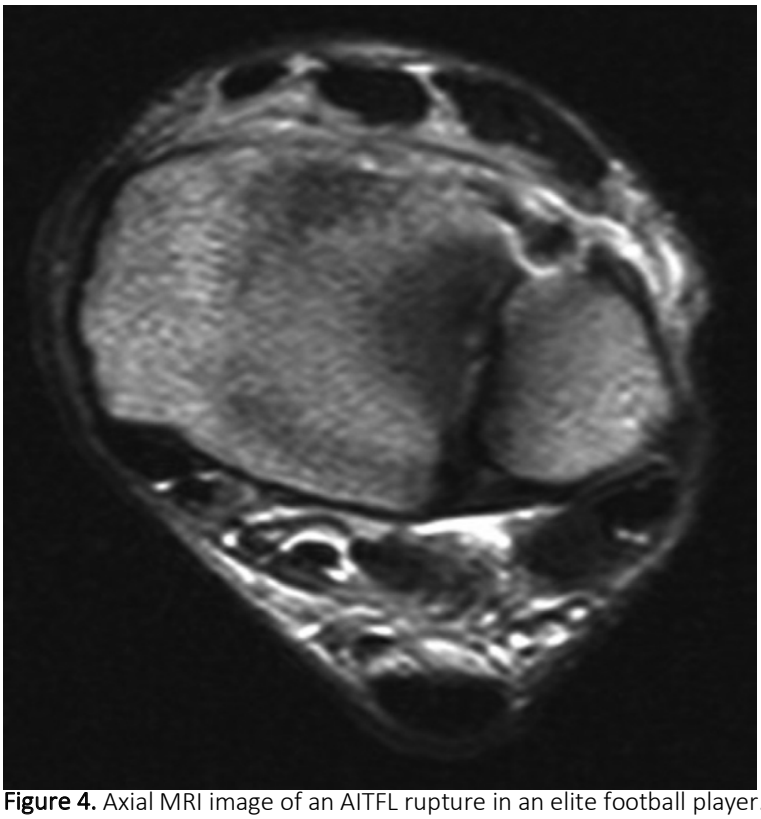

Figure 4. Axial MRI image of an AITFL rupture in an elite football player.

A force of 100 N has been stated as sufficient, and tibiofibular clear space widening exceeding 5 mm in the case of an unstable syndesmosis will require stabilization.[59] Whenever in doubt about syndesmotic instability (Figure 4), stabilization should be performed because of the long-term complications caused by chronic syndesmotic instability.[59]

RETURN TO PLAY and PREVENTION

"Low" Ankle Sprain

It is difficult to determine when an athlete can return to play (RTP) following an ankle sprain. Residual disability of ankle sprains is often caused by inadequate proprioceptive rehabilitation and a potentially overly hurried RTP.[47] Self-reported ankle scoring systems (FAOS[60]) are not validated for RTP decisions, but can be useful to evaluate the effectiveness of the rehabilitation protocol. Use of functional performance tests to assess an athlete's ability to perform sport-specific skills is considered helpful.[46] Tests can progress from the single-legged balance test[61] to more complex tests, such as the Star Excursion Balance Test[62], the Y-balance test[63], and the agility t-test.[64] The rehabilitation process should

never abruptly be stopped, and continuing sport-specific rehabilitation will help to minimize the risk of deficits or re-injuries. The time needed to RTP in lateral ligamentous injury will depend upon several factors, including severity of the initial injury, the patient's ability and the rehabilitation facilities available and ranges from 10 days to 6 weeks.

The most important risk factor for developing a chronic ankle sprain is a previous ankle sprain. This is probably due to reduced proprioceptive function and deficient mechanical stability. There is academic evidence that neuromuscular training, especially balance and proprioceptive training, is effective for the prevention of recurrent ankle sprains. This form of therapy can also be effectively performed at home.[65]

"High" Ankle Sprain

Athletes who sustain a syndesmotic ankle sprain typically should go through much longer recovery periods than those who sustain a lateral ankle sprain.[13] RTP in Grade I injuries is usually at 6-8 weeks' post-injury, but is variable. Professional athletes with stable isolated Grade II syndesmotic injuries are reported to RTP at a mean of 45 days, compared with 64 days for those with unstable Grade II injuries.[54] Also, athletes with injury to both the AITFL and deltoid ligament took longer to RTP than those with an AITFL injury alone, and IOL injury on MRI and PITFL injury on MRI were both independently associated with a delay in RTP.[54]
In the case of surgically treated Grade III injuries, the expected time frame to RTP is between 10 and 14 weeks[13,55], although RTP as early as 6 weeks has been described in case series.[66]

RTP in syndesmotic injury is permitted when able to single- leg hop for 30 seconds without significant pain.[59] To our knowledge, there are no specific studies on prevention of syndesmotic re-injury. Although it might be assumed that neuromuscular bracing and bracing or taping is beneficial, injury mechanisms differ and further investigation is required to increase our understanding of syndesmotic injuries and improve treatment and prevention of this significant injury.[13]

CONCLUSION

"Low" and "high" ankle sprains in athletes are very different entities in the mechanism of injury, clinical features, diagnostic setup, management, and prevention. The aim of this review is to document the specific characteristics of both and present the best evidence-based literature data along. If proper management can be started after early detection, excellent results can be obtained in both types of ankle sprains. This is not the case for the evolution to chronic instabilities and combined injuries in both and this needs to be avoided at all times. Therefore, further research is needed to fine tune the preventative strategies and treatment in both types of athlete ankle sprains.

"Low" Ankle Sprain Factbox

- Physical examination for the detection and classification of lateral ankle ligaments is best delayed for (4-5 days) after initial trauma to give better results, knowing that the Ottawa rules remain valuable in the acute setting.
- Most acute lateral ligament injuries can be treated conservatively with adequate rehabilitation.
- Surgery might be considered in professional athletes with acute Grade III injuries, as it may provide lower incidence of chronic ankle instability than conservative treatment.
- RTP should include functional performance tests.

"High Ankle Sprain" Factbox

- Syndesmotic injury generally occurs in association with other injuries, especially fractures.
- Stable syndesmotic injuries (types I and IIa) should be treated conservatively, whereas unstable injuries (types IIb and III) require surgical fixation.
- RTP is generally prolonged in syndesmotic injury and allowed when able to single-leg hop for 30 seconds.

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Concomitant injuries in chronic ankle instability.

Clinical Research on Foot and Ankle Journal (2018)

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Concomitant Injuries in Chronic Ankle Instability
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Clin Res Foot Ankle 2018, 6:3
DOI: 10.4172/2329-910X.1000281

ABSTRACT

Chronic ankle instability is associated with a number of coincident injuries about the ankle joint. Improvements in ankle arthroscopy and preoperative imaging modalities have aided in the identification and treatment of these lesions. Although their clinical significance may be variable, the presence of concomitant lesions in chronic ankle instability predisposes patients to chronic pain and osteoarthritis. A comprehensive review of the literature reveals that a multitude of studies have described associated lesions in patients with chronic ankle instability.

These lesions include peroneal tendon injuries, chondral and osteochondral lesions of the tibial plafond and talar dome, intra-articular loose bodies, anterior/anterolateral ankle soft tissue impingement, lateral malleolus ossicles, tibiofibular syndesmosis injuries, and peroneal nerve injuries. This review serves as a comprehensive analysis of the literature, focusing on identification, treatment, and long-term outcomes of concomitant injuries in chronic ankle instability.

INTRODUCTION

Chronic ankle instability (CAI) occurs in individuals with a history of at least one prior ankle sprain resulting in chronic laxity of the ligaments surrounding the ankle joint[1,2]. Lateral ankle sprains are the most common injury in sports, resulting typically from excessive inversion, plantarflexion, and internal rotation of the ankle[3]. Lateral ankle sprains are most commonly due to injury to the anterior talofibular ligament (ATFL), and less commonly the calcaneofibular ligament (CFL) or posterior talofibular ligament (PTFL) which completes the lateral ankle ligament complex. Injuries to the deltoid ligament and subsequent medial ankle instability are rare[4].

Due to the frequency of the injury and the perception of ankle sprains as being minor injuries, a majority of patients with an acute ankle sprain do not seek medical care which may lead to increased residual symptoms such as chronic ankle instability[1,5]. Ankle instability can be described as mechanical instability (pathologic laxity of the ligaments in the ankle) or functional instability (insufficiency in proprioception/ neuromuscular control) [6,7].

Chronic ankle instability has been estimated to occur in 10 to 20 percent of patients who sustain an acute ankle sprain[8]. A majority of patients with chronic ankle instability continue to have instability with conservative management and

a small subset will have chronic pain and progress with osteoarthritis of the ankle joint[9]. Advances in lateral ankle ligament reconstruction in the past 50 years, specifically with the Broström and Broström-Gould techniques, have improved clinical results with surgery[10-12] (Figure 1).

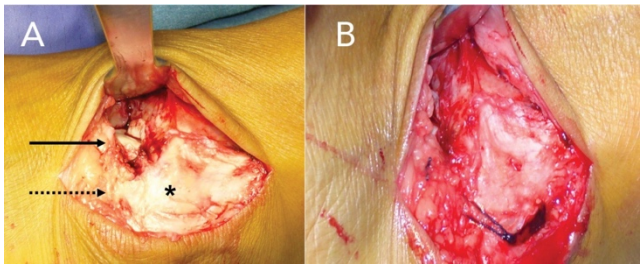

Figure 1: ATFL tear and repair with Brostrom-Gould reconstruction. Shows intra-operative photographs of (A) an ATFL tear, and (B) a lateral ligament reconstruction with Brostrom-Gould technique.

However, 13 to 35 percent of patients have persistent pain and symptoms after a successful lateral ligament reconstruction which some attribute to concomitant intraarticular lesions[13,14]. Improvements in preoperative imaging modalities, specifically magnetic resonance imaging (MRI), have aided in more accurately identifying these lesions prior to surgery. Prior to the rise in prevalence of ankle arthroscopy, intra-articular lesions were evaluated through an open ankle arthrotomy which has limited visualization and increased morbidity compared to arthroscopy.

The ability to identify and treat concomitant lesions in chronic ankle instability has improved, and their role in functional outcomes are significant. The most frequently encountered lesions include peroneal tendon injuries, osteochondral lesions of the tibia and talus, chondral injuries and chondromalacia, intra-articular loose bodies, anterior and anterolateral ankle impingement, lateral malleolus ossicles, syndesmosis widening, and peroneal nerve injuries.

A majority of the current literature looking at concomitant injuries in chronic ankle instability is primarily retrospective case series and cohorts. The prevalent studies are highlighted in detail in the following manuscript. To our knowledge, this is the first comprehensive review of the literature highlighting the diagnosis, frequency, clinical significance, and treatments for all injuries associated with chronic ankle instability.

REVIEW

Diagnosis

One of the first large case series to study associated injuries in patients with CAI was by DiGiovanni, et al. which was a retrospective review of 61 patients with chronic ankle instability that underwent lateral ligament reconstruction and open ankle arthrotomy. The authors found a number of associated injuries about the ankle joint.
These injuries and their frequencies included: peroneal tenosynovitis (47/61, 77%), anterolateral impingement lesion (41/61, 67%), attenuated peroneal retinaculum (33/61, 54%), ankle synovitis (30/61, 49%), loose bodies (16/61, 26%), peroneus brevis tear (15/61, 25%), osteochondral lesion of the talus (14/61, 23%), ATFL avulsion (7/61, 11%), accessory peroneus quartus muscle (5/61, 8%), medial ankle tendon tenosynovitis (3/61, 5%), and capsular avulsion fracture (2/61, 3%). One hundred percent of patients had at least one associated injury[15]. This study demonstrates that a variety of concomitant injuries are associated with CAI, and they are present in the vast majority of patients.

Similarly, Strauss, et al. looked at 180 ankles in 160 patients undergoing modified Brostrom-Gould lateral ankle ligament reconstruction for chronic ankle instability. In this cohort, the authors reviewed physical examination, clinical history, radiographs, and intraoperative findings to find the incidence of concomitant extra- articular lesions in CAI. They found that 64% of all patients had associated extra-articular conditions. These included peroneal tendon injuries (51/180, 28%), os trigonum lesions (23/180, 13%), lateral gutter ossicles (18/180, 10%), hindfoot varus malalignment (15/180, 8%), anterior tibial spurs (5/180, 3%), and tarsal coalitions (3/180, 2%)[16].

This study reported lower incidence of concomitant injuries, but there was no mention of intraarticular assessment with arthrotomy or arthroscopy. In addition to these injuries, anterior process fractures of the calcaneus can occur in conjunction with acute ankle sprains, and are at risk of misdiagnosis[17].

Magnetic resonance imaging

Diagnosis of concomitant lesion in chronic ankle instability was difficult prior to the increased utilization of MRI and arthroscopy. Improvements in MRI have allowed for more accurate diagnoses of lesions about the foot and ankle and help

in the work up of chronic ankle pain. O'Neill, et al. found that preoperative MRI radiology reports identified 39% (15/38) of chondral injuries, 56% (10/18) of peroneal tears, and 57% (4/7) of loose bodies, with an overall sensitivity of 45% for detecting lesions that were visualized intraoperatively[18]. Additionally, the attending surgeon's review of the MRI's yielded identification of 47% (18/38) of chondral injuries, 89% (16/18) of peroneal tears, and 71% (5/7) of loose bodies with an overall sensitivity of 63%.

The conclusion of the study was that while having the surgeon review the MRI improves preoperative diagnosis of concomitant injuries, MRI may not be adequate to detect lesions before surgery[18].

Cha et al. similarly compared preoperative MRI findings to intraoperative arthroscopy in patients who had surgery for chronic lateral ankle instability. The sensitivities for MRI in detecting each individual lesion were 60% for ATFL injury, 46% for osteochondral lesions of the talus (OLT), 21% for syndesmosis injury, 21% for synovitis, and 22% for anterior osteophyte impingement [19].
The low sensitivity and interobserver reliability of preoperative MRI tests prompted the authors of this study to strongly recommend arthroscopic examination in all cases to identify and treat concomitant injuries[19].

Arthroscopy

A multitude of case series and retrospective cohorts are present in the literature looking at the use of arthroscopy for diagnosis of concomitant injuries in chronic ankle instability. Ankle arthroscopy has increased the ability to accurately identify intraarticular ankle pathology while maintaining low long-term morbidity to the patient[20]. The general consensus is that Intra-articular lesions associated with chronic ankle instability are common, and ankle arthroscopy is currently the gold standard in diagnosing the injuries.
Schafer et al. studied 110 consecutive patients prospectively with chronic ankle instability who underwent ankle arthroscopy. Arthroscopy identified 42 patients (38%) had synovitis, 7 (6%) had ventral scarring, 11 (10%) had a synovial plica, 8 (7%) had syndesmosis injury, 59 (54%) had talar cartilage injuries, and 19 (17%) had distal tibial cartilage injuries.
The ligamentous injuries included rupture of the ATFL in 64%, rupture of CFL in 41%, and deltoid ligament rupture in 6%[21]. Choi et al. looked at the arthroscopic findings in patients undergoing modified Broström procedure for chronic lateral ankle instability. This case series found that 97% (63/65) of ankles had associated intra-articular lesions. They included syndesmotic widening (19/65, 29%),

osteochondral lesion (15/65, 23%), lateral malleolus ossicles (25/65, 38%), soft tissue impingement (53/65, 82%), and osteophyte formation (7/65, 11%).

The authors found that the postoperative outcome scores negatively correlated with the number of lesions, and syndesmotic widening, osteochondral lesions, and ossicles were independently predictors of poor outcome.
Osteophyte formation and soft tissue impingement had no significant correlation[13]. Similarly, Hua, et al. found intra-articular lesions in 91% (79/87) of ankles undergoing arthroscopic evaluation prior to modified Broström procedure in a retrospective review.
The lesions included synovitis/soft tissue impingement (75/87, 86%), chondral injury (33/87, 38%), anterior tibial osteophyte (23/87, 26%), loose bodies (7/87, 8%), and syndesmotic injury (6/87, 7%). Outcome scores at least 12 months postoperatively were significantly higher in patients without chondral lesions[22].

A number of additional case series have been performed looking at the incidence and outcomes of patients with concomitant intra-articular lesions in the setting of chronic ankle instability and are included in Table 1.

Study(N)	Soft tissue impingement	Synovitis	Syndesmosis injury	Osteochondral lesions	Chondral injury	Ossicles	Osteophytes	Loose bodies
Choi [13] (65)	82%	NR	29%	23%	NR	39%	11%	NR
Schafer [21] (110)	NR	38%	7%	NR	54%	NR	NR	NR
Hua[22] (87)	86%	NR	6.90%	NR	38%	NR	26%	8.00%
Ferkel [20] (21)	NR	76%	NR	19%	33%	29%	19%	24%
Lee [23] (28)	14%	100%	NR	7.10%	39%	NR	14%	11%
Liszka [24] (25)	NR	75%	NR	NR	56%	NR	52%	20%
Hintermann [25] (148)	17%	32%	8.80%	NR	54%	NR	12%	NR
Komenda [26] (55)	NR	69%	NR	16%	22%	25%	11%	22%
Odak [27] (100)	NR	63%	NR	17%	NR	NR	12%	1.00%

Table 1: Incidence of intra-articular lesions in patients with chronic ankle instability found on arthroscopy.

The overall incidence of associated intra-articular lesions is as high as 100 percent in several studies. Additionally, these studies identified the following associated intra-articular lesions with arthroscopy: Synovitis (range of incidence 32%-100%), soft tissue impingement (14%-86%), chondral injury (22%-56%),

osteochondral lesion (7%-23%), lateral malleolus ossicle (25%-39%), anterior osteophyte (11%-52%), and intraarticular loose body (1%-24%)[12,22-28].

What can be concluded from these studies is that chronic ankle instability is associated with an extremely high prevalence of associated intra-articular pathology. In the hands of many surgeons, this evidence shows that arthroscopic evaluation of the ankle joint during lateral ligament reconstruction is indicated in all cases to identify intra-articular pathology. Preoperative imaging and examination may identify some associated pathology, but a significant number of intra-articular lesions will be missed without arthroscopic evaluation of the joint.

SPECIFIC LESIONS: DIAGNOSIS and MANAGEMENT

Peroneal tendon injuries

Peroneal tendon injuries, specifically peroneus brevis, are commonly seen in association with chronic ankle instability (Figure 2). The proposed mechanism is that laxity in the superior peroneal retinaculum allows for anterior slipping of the peroneus brevis tendon in the retrofibular groove. This causes the tendon to ride over the sharp posterior edge of the fibula, leading to longitudinal tears in the central tendon[29].
The incidence of peroneal tendon pathology in the setting of CAI is relatively common. A cadaveric study found an incidence of peroneal tears in 11% (14/124) of ankles with unknown history; however, the clinical relevance of this number is unknown[30]. A clinical retrospective study found that 38% (31/82) of ankles operated on with chronic lateral ankle instability had peroneal tendinopathy and 13% (11/82) had frank peroneal tendon tears[31].

Most patients with a peroneal tendon injury will complain of retromalleolar pain and tenderness, and may present with clinical subluxation of the peroneal tendons with ankle circumduction.
MRI is a useful tool in assisting with the diagnosis of peroneal tendinopathy with a sensitivity of 84% and specificity of 75%[31]. In assessing for peroneal tendinopathy in patients with CAI, one should utilize clinical history, physical examination, and imaging studies as well as thorough intraoperative assessment.

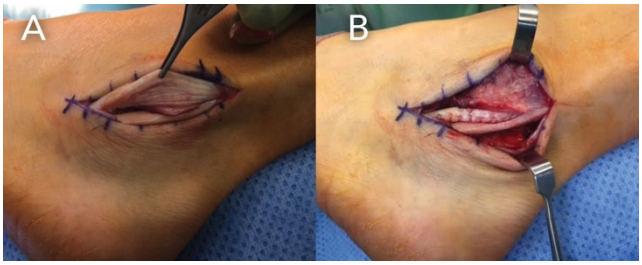

Figure 2: Peroneal tendon tear and repair. Shows intra-operative photographs of (A) a longitudinal peroneal tendon tear, and (B) side to side repair.

Conservative management of peroneal tendon tears is an option; however, in the setting of chronic lateral ankle instability and chronic ankle pain, operative management may be considered.

Operative management with a simple debridement of the diseased split tendon and side to side repair is appropriate in cases with less than 50% tendon involvement and has been described with 90% good to excellent results[32,33]. In cases with greater than 50% tendon involvement, the surgeon must consider allograft augmentation or lateral ligament reconstruction utilizing the peroneus brevis tendon such as in a Chrisman-Snook procedure[34].

Osteochondral/chondral injuries

One of the most classically recognized lesions seen with chronic ankle instability is in the spectrum of chondral and osteochondral lesions of the talus (OLT) and the tibial plafond (Figure 3). These intra-articular lesions are a common source of pain in chronic ankle instability, and may be a precursor to posttraumatic ankle arthritis. The incidence of chondral injuries in CAI varies in the literature greatly from 22% to 95%.

Osteochondral lesions are diagnosed less frequently in CAI with an incidence of 7% to 23% in the literature[13,23]. Taga et al. looked at 31 ankles with lateral ligament injury and found that eight out of nine freshly injured and 21 of 22 chronic injured ankles had cartilage injuries. The most common area of injury was the anteromedial edge of the tibial plafond. Chondral injuries greater than one half the thickness of the articular cartilage were only found in chronic instability patients, leading the authors to suggest that chondral injuries increase in severity over time[35]. In a more recent study by Sugimoto et al., cartilage injuries were graded in patients with CAI. The group found 23% (23/99) with normal cartilage,

35% (35/99) with grade one chondromalacia, 24% (24/99) with grade two, and 17% (17/99) with grade three. They also found a significant association between worse cartilage injury and increased patient age and talar tilt angle[36].
Deltoid ligament insufficiency is associated with a significantly higher rate of chondral injuries in patients compared to isolated lateral ankle instability[20,24].

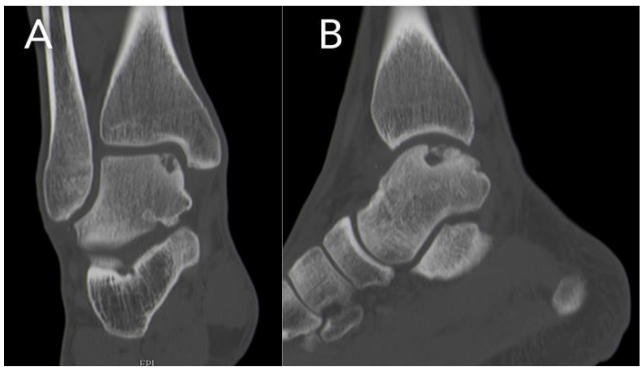

Figure 3: Osteochondral lesion of the talus. Shows (A) coronal and (B) sagittal cuts of an ankle computed tomography scan demonstrating medial sided taller dome osteochondral lesion.

When assessing medial versus lateral osteochondral lesions in the talus, it has been shown that lateral talar are nearly always traumatic in nature, while medial lesions are not necessarily. Furthermore, medial lesions typically present later, have more cystic changes, and have worse clinical outcomes than lateral lesions[37].
Non-displaced osteochondral lesions can often be treated conservatively, although this is more successful in the pediatric population. Smaller lesions, typically less than 15 mm in diameter, respond well to bone marrow stimulation techniques such as microfracture drilling.
Large cystic lesions may require more aggressive techniques such as osteochondral autograft or allografts, autologous chondrocyte implantation, and matrix- associated chondrocyte implantation[38].

The results of specific treatment techniques for osteochondral and chondral lesions in the ankle joint are out of the scope of this review; however, it has been shown that outcomes for OLTs in patients with chronic ankle instability are significantly worse than patients without CAI[39]. Unfortunately, the natural course for many patients with chronic ankle instability is worsening chondromalacia leading to posttraumatic osteoarthritis[40].

Lateral malleolus ossicles

The presence of ossicles adjacent to the lateral malleolus in the setting of chronic lateral ankle instability are presumed to be from an avulsion of the tip of the lateral malleolus from pull of the CFL or ATFL most commonly (Figure 4). Ossicles typically are asymptomatic, but may be painful secondary to stress in the connection of the ossicle to the fibular tip[41]. The incidence of lateral malleolus ossicles in patients with CAI ranges from 25% to 38% in the literature.

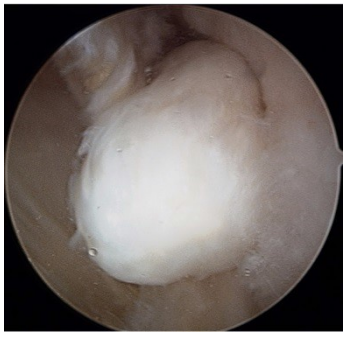

Figure 4: Lateral malleolus ossicle. It is an arthroscopic, intra-operative image of a lateral malleolus ossicle in a patient with chronic lateral ankle instability.

Treatment in patients with symptomatic lateral malleolus ossicles is typically with ossicle resection and lateral ligament reconstruction. With a larger sized ossicle (greater than 10 mm), fibrous union takedown and ossicle fusion to fibular tip should be considered to prevent persistent anterior talar displacement[42].

The clinical significance of lateral malleolar ossicles in patients with CAI is mixed in the literature. Choi, et al. found that the presence of lateral malleolar ossicles independently predicted unsatisfactory results in patients undergoing lateral ligament reconstruction[13]. However, two other retrospective reviews showed no difference in clinical results when comparing patients with ossicle resection plus modified Broström procedure to patients with no ossicle at all[43,44].

Anterior ankle impingement

Anterior impingement is a common source of ankle pain, and is believed to be a direct consequence of chronic ankle instability in certain cases[45]. One study compared patients who underwent lateral ligament reconstruction to a cohort of

normal ankles and found a 3.37 times increased incidence of anterior impingement spurs in the lateral ligament reconstruction group[46].

Anterior ankle impingement can be from osteophytes or soft tissue impingement and is seen clinically with restricted ankle dorsiflexion and anterior ankle pain. Osteophytes are present in 11% to 26% of chronic ankle instability cases according to the literature.
Soft tissue impingement has a much broader incidence between studies, ranging between 14% and 82%. Bassett lesions can develop in CAI as a thickened cord in the anteroinferior tibiofibular ligament in response to recurrent ankle instability events and contribute to impingement as well[47].

The diagnosis of anterior or anterolateral ankle impingement is typically with clinical examination, but MRI is a useful tool in diagnosis with 91.9% sensitivity and 84.4% specificity (Figure 5)[48].
Management of anterior impingement initially with conservative treatment may include activity modification, anti-inflammatory medications, physical therapy, bracing, and injections. With failure of conservative measures, operative intervention can be considered.

The majority of anterior ankle impingement currently is treated with arthroscopic debridement. Recent studies have shown 90% to 97% good and excellent postoperative results with arthroscopic debridement of anterior ankle impingement in the setting of prior ankle instability event[49,50].

Ankle syndesmotic injuries can be associated with significant pain and morbidity in the ankle and often times are missed at the time of initial presentation of ankle sprains. A concomitant syndesmotic injury in a patient with CAI is an independent predictor of poor outcome[13]. A study by Takao et al. looked at different diagnostic tools in assessing for tibiofibular syndesmosis injuries.

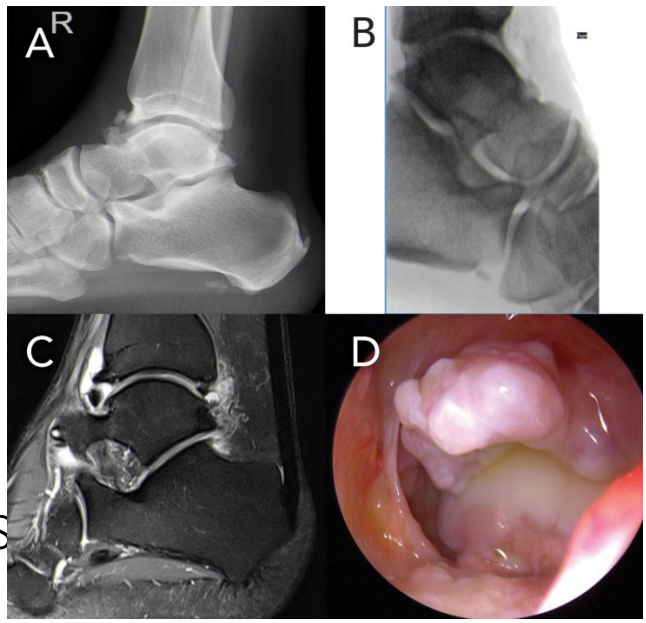

Figure 5: Anterior ankle impingement. (A) a pre-operative lateral radiograph of a patient with bony anterior ankle impingement from an osteophyte, and (B) intra-operative fluoroscopy image after resection of the osteophyte. (C) A sagittal MRI cut demonstrating another example of anterior bony ankle impingement. (D) Intra-operative arthroscopic view of an anterior ankle osteophyte causing impingement.

The authors found that MRI was significantly better at identifying syndesmosis injuries (sensitivity 100%, specificity 93.1% for anterior inferior tibiofibular ligament injury and 100%, 100% for posterior inferior tibiofibular ligament injury) compared to AP radiographs (44.1%, 100%) and mortise radiographs (58.3%, 100%)[51] (Figure 6).

In the chronic syndesmosis disruption, treatment with arthroscopic debridement alone typically is sufficient in providing pain relief, improved stiffness, and better ankle stability[52].

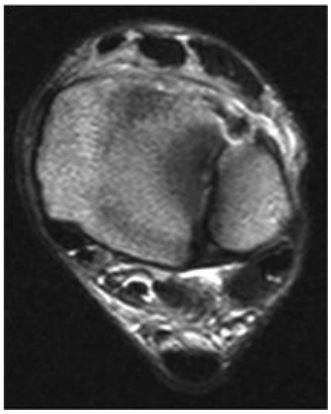

Figure 6: Ankle syndesmotic injury. An axial MRI sequence of an individual with a tibiofibular syndesmotic injury characterized by an AITFL rupture.

Peroneal Nerve Injury

Peroneal nerve injuries after ankle sprain are rare, but there are a number of case reports in the literature describing this injury. In the vast majority of cases, this occurs as a neuropraxic type injury at the time of ankle sprain and resolved with conservative management[53].
The insufficiency of the lateral ankle ligaments causes increased strain in the peroneal nerve. Although a rare injury, a case report described chronic peroneal nerve injury in the setting of recurrent lateral ankle instability events[54].

CONCLUSION

Concomitant injuries in chronic ankle instability are common sources of pain and morbidity for patients. Some of the most common injuries include osteochondral and chondral lesions, peroneal tendinopathy, syndesmotic injury, lateral malleolar ossicles, soft tissue and osteophyte impingement, and peroneal nerve injury.
The incidence of these injuries is higher than many practitioners believe. Even with a thorough history and physical examination as well as cross-sectional imaging with MRI, a significant amount of these injuries are still missed pre-operatively. The morbidity of missed concomitant injuries in chronic ankle instability is difficult to assess, and many surgeons recommend arthroscopic evaluation in all cases of ankle ligament reconstruction. Management of each individual lesion is different; therefore, one must be diligent in operative preparation for chronic ankle instability cases.

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CHAPTER

3

BIOMECHANICAL CADAVERIC LATERAL ANKLE LIGAMENT LABORATORY TESTING USING 3 TESTING PROTOCOLS

CHAPTER 3:
Biomechanical cadaveric lateral ankle ligament laboratory testing using 3 testing protocols

The Role of Calcaneofibular Ligament (CFL) Injury in Ankle Instability: Implications for Surgical Management.

American Journal of Sports Medicine (2018)

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The Role of Calcaneofibular Ligament Injury in Ankle Instability:

Implications for Surgical Management.

Hunt KJ, Pereira H, Kelley J, Anderson N, Fuld R, Baldini T, Kumparatana P, D'Hooghe P.
Am J Sports Med. 2019 Feb;47(2):431-437. doi: 10.1177/0363546518815160. Epub 2018 Dec 20.

ABSTRACT

Background: Acute inversion ankle sprains are among the most common musculoskeletal injuries. Higher Grade sprains, including anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) injury, can be particularly challenging. The precise impact of CFL injury in ankle instability is unclear.

Purpose: We hypothesized that CFL injury will result in decreased stiffness, peak torque, and increased talus and calcaneus motion, as well as alter ankle contact mechanics when compared to the uninjured ankle and the ATFL only injured ankle in a cadaveric model.

Study Design: Controlled Laboratory Study

Methods: Ten matched-pairs of cadaver specimens with a pressure sensor in the ankle joint and motion trackers on the fibula, talus, and calcaneus were mounted on an Instron with 20° of ankle plantar flexion and 15° of internal rotation. Intact specimens were axially loaded to body weight, then underwent inversion along the anatomic axis of the ankle from 0° to 20°. The ATFL and
CFL were sequentially sectioned and underwent inversion testing for each condition. Linear mixed models (LMMs) were used to determine significance for stiffness, peak torque, peak pressure, contact area, and inversion angles of the talus and calcaneus, relative to the fibula across the three conditions.

Results: Stiffness and peak torque did not significantly decrease after sectioning the ATFL, but decreased significantly after sectioning the CFL. Peak pressures in the tibiotalar joint decreased and mean contact area increased significantly following CFL release. There was significantly more inversion of the talus and calcaneus as well as calcaneus medial displacement with weight-
bearing inversion after sectioning the CFL.

Conclusions: The CFL contributes considerably to lateral ankle instability. Higher Grade sprains that include CFL injury result in significant decreases in rotation stiffness, peak torque, substantial alteration of contact mechanics at the ankle joint, increased inversion of the talus and calcaneus, and increased medial displacement of the calcaneus.

Clinical Relevance: Repair of the CFL should be considered during lateral ligament reconstruction when injured, and there may be a role for early repair in high-

grade injuries to avoid intermediate and long-term consequences of a loose or incompetent CFL.

What is known about the subject?
The ATFL and CFL are both important lateral ankle stabilizers in internal rotation and inversion. While there is a trend towards worse outcomes in combined ATFL and CFL injuries, there is still a lack of knowledge concerning the implications of insufficiency of the CFL as well as the possible relevance of its respective repair. Additionally, there is no current consensus amongst the Orthopaedic community whether the CFL should be repaired in high Grade ankle sprains. Hence, biomechanical studies, particularly in weight-bearing conditions are highly required.

What this study adds to existing knowledge?
This study presents the first biomechanical study examining the influence of the ATFL and CFL during weight-bearing inversion injury conditions concerning both joint stability and kinematics.
Sequentially greater inversion of the talus and calcaneus was noticed with progressive ligament injury (ATFL alone followed by combined ATFL and CFL insufficiency).
This study suggests that the CFL plays a more significant role in ankle joint stability and contact mechanics when compared to the ATFL, and that repair of the CFL should be considered during lateral ligament reconstruction. A CFL-deficient ankle has significantly different joint mechanics than the intact ankle, and there may be an important role for early repair of the CFL in high Grade ankle sprains.

INTRODUCTION

Acute inversion ankle sprains are among the most common musculoskeletal injuries in both athletes and non-athletes. The incidence in the United States is 30,000 ankle sprains/day and accounts for 7-10% of emergency room visits.[4, 8, 9] It is estimated that 25-40% of all sports-related injuries involve the ankle.[8, 15] Non-operative management of acute ankle sprains is appropriate for the majority of ankle sprains. However, it is estimated that 20% of severe ankle sprains will lead to chronic ankle instability, diminished athletic performance, and further joint injuries.[20] Inversion force of the ankle with the foot in plantarflexion is the most common mechanism of ankle ligament injury.[13] Two of the most important ligaments in the ankle's lateral

ligament complex during acute lateral ankle injury are the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL). The ATFL is responsible for restricting internal rotation of the talus in the mortise and inversion during plantar flexion. The ATFL is the most often injured ligament in acute ankle sprains with a failure load at around 138 N, which is reported to be 2 to 3.5 times lower than the failure of the CFL.[2, 19, 29, 30]

In a cadaver model, Bahr et al. measured the maximum force in the ATFL to be 76±23 N and the highest load in the CFL to be 109±28 N in a cadaver model.[3] This ATFL load is 55% of the 138 N failure load and the CFL is 22% to 39% of this failure load. High Grade ankle sprains include both the ATFL and CFL. The CFL is nearly exclusively responsible for resistance to inversion during dorsiflexion in the neutral state. During plantarflexion, the CFL resists inversion alongside the ATFL, and also acts as a stabilizer of the subtalar joint.[16] In an estimated 50-70% of high grade ankle sprains, it is thought that following ATFL elongation, the stronger CFL becomes stretched until it fails at around 345 N.[2, 12]

For patients who fail conservative management for high-grade sprains, the gold standard surgical procedure is the lateral ligament repair first described by Broström.[6]

Recently, arthroscopic techniques to repair the ATFL have emerged as clinically effective in the short term.[26] The impact of CFL injury in ankle instability is unclear and there is variability in current practices in terms of whether the CFL is repaired during lateral ligament repair. For example, some surgeons suggest that repair of the CFL is unnecessary, yet a survey of an international consensus group indicates that 80% of respondents routinely repair the CFL during a lateral ligament repair procedure.[1, 23] Some authors do not advocate repairing the CFL based on biomechanical data and clinical outcomes data.[21, 22]

Contributing to the lack of consensus on the necessity of repairing the CFL are limited biomechanical data in the literature examining what role the CFL plays in lateral ankle stability. The objective of this study was to evaluate the impact of CFL injury on ankle joint stability and biomechanics. We hypothesized that CFL injury will result in decreased stiffness, decreased peak torque, and increased talus and calcaneus motion, as well as alteration of ankle contact mechanics when compared to the uninjured ankle and the ATFL only injured ankle in a cadaveric model.

METHODS

Ten matched pairs of fresh frozen human cadaveric specimens from mid-tibia to toe tip, (5 male, average age 51.4 years, range 38-60; 5 female, average age 53.8 years, range 32-64) were obtained for experimentation from a tissue bank. This project followed all Institutional Review Board requirements in our institution for cadaver laboratory research. Previous studies have established the use of fresh frozen specimens compared to specimens not frozen, as there was little effect on the gross biomechanical properties of the ligaments and other connective tissues due to freezing.[25, 31]

Each specimen was transected at the mid-shaft tibia/fibula. All specimens were evaluated visually and radiographically for signs of gross deformity, previous operation, fracture, and rheumatoid arthritis. Specimens were wrapped in moist gauze and placed in a -20°C freezer for storage.
The specimens were thawed at room temperature on the day they were prepared and tested. The proximal 4" of soft tissue was removed from the tibia and fibula. The fibula was rigidly fixed to the tibia with a 4.5 mm cortex screw. The proximal 3" of the tibia/fibula was potted with an epoxy (SmoothCast 321; Smooth-On, Inc., Easton, PA, USA) in a 3" diameter round tube. To facilitate approach to the tibiotalar joint, the extensor digitorum longus, tibialis anterior, extensor hallucis longus, and Achilles tendons were sectioned.[17]
The plantar surface was secured in an epoxy bed with one additional screw for fixation in the calcaneus. The skin and soft tissue covering the ATFL and CFL were carefully removed without damaging either ligament. Biomechanical testing was performed on a material testing system (Instron Model 1321 109 with 8500 controllers; Instron Corporation, Norwood, MA, USA). A 3D, 2 camera motion capture system (Innovision Systems Inc., Columbiaville, MI, USA) was used with custom reflective trackers each rigidly attached with two, 3.0 mm pins, to the fibula, talus, and calcaneus to record the motion of each bone during testing.

A pressure measurement system (Model 5033 sensors; Tekscan Inc., Boston, MA, USA) was used to obtain intra-articular tibiotalar pressure data. The sensor was coated with petroleum jelly before being inserted into the ankle joint to minimize the shear forces on the sensor. The pressure sensor is 38.4mm 115 long and 26.7mm wide. It contains 46 rows and 32 columns of 0.694 mm^2 sensels for a total of 1472 sensels. The sensor was inserted so that there were uncontacted sensels anterior, posterior, and lateral to the initial points of contact present on the sensor reading. In many cases, the medial edge of the

sensor abutted the bony medial border of the joint. To calibrate the sensors, they were conditioned for 4 cycles to 1800 N, followed by a 10-point power law calibration. Conditioning and calibration cycles consisted of loading for 10 seconds, held at designated load for 30 seconds, unloaded over 10 seconds, and recovery for 2 minutes.[24]

Each specimen was mounted with the tibia horizontal onto the testing apparatus in 20° of plantarflexion and 15° of internal rotation, ensuring that the center of rotation of the tibiotalar joint was aligned with the rotation of axis of the actuator.[7, 14] The tibia was fixed to a platform on the base of the material testing system that was mounted on two linear bearings that allowed free motion in the anatomic superior/inferior direction.

Specimens were axially loaded in compression to full body weight by running a cable horizontally from the platform that the tibia was fixed to over a pulley. Weights were hung on the cable equal to the body weight of each individual donor that was obtained from their donor summary report.

Each ankle was preconditioned for 10 cycles from 0° to 10° of inversion at 0.25 Hz.[29] After preconditioning, a pressure sensor was inserted into the tibiotalar joint posteriorly to avoid crimping of the sensor (Figure 1A, 1B). Each ankle was tested from 0° to 20° of inversion along the anatomic axis of the ankle at a rate of 5°/s for three cycles.

The ATFL and CFL were then sequentially sectioned, and inversion testing was repeated for each of the following conditions:

(1) intact;

(2) ATFL-injury sectioning;

(3) CFL-injury sectioning.

Data were collected at 25 Hz on a PC equipped with an analog to digital board and data acquisition software.[7]

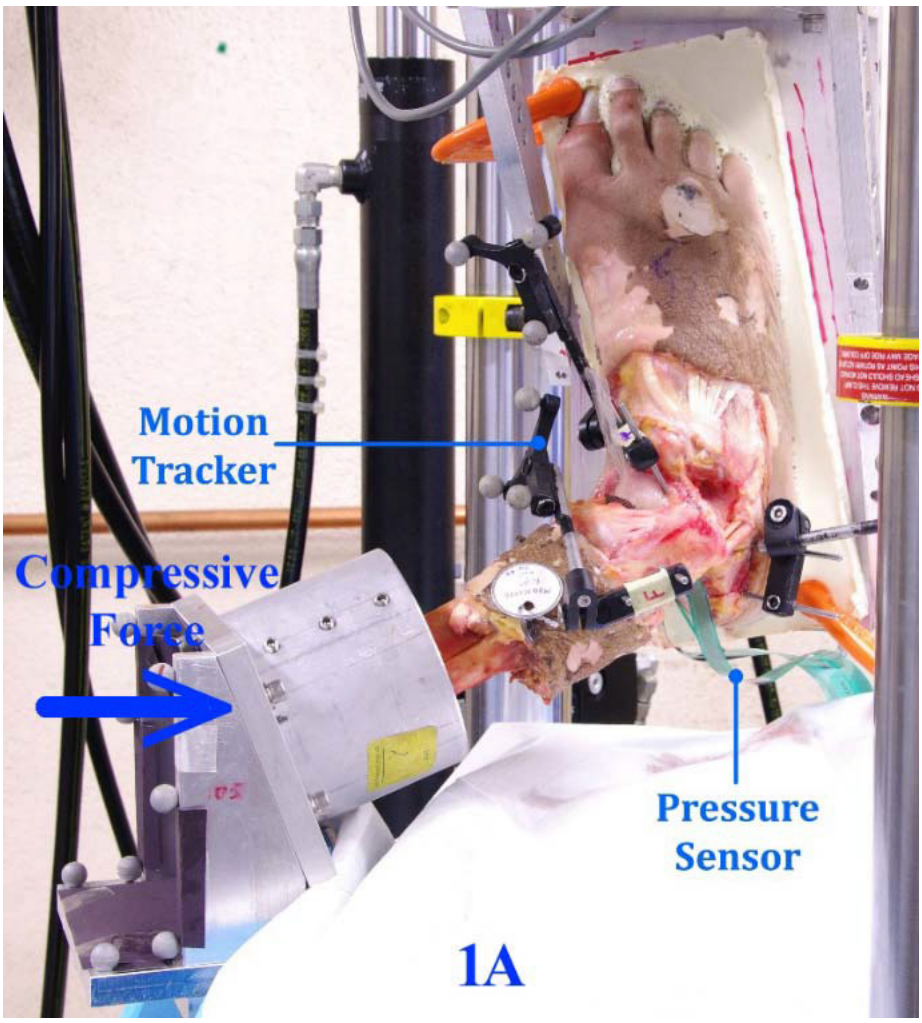

Figure 1(A). Test Setup showing the ankle is in 20° of plantar flexion and internally rotated 15°. The platform the tibia is mounted to sits on linear bearings that allow free motion in the anatomic superior/inferior direction (horizontal in the figure).
The cable that applies the axial compression force cannot be seen in the picture but it runs horizontally to the right of the picture where it runs over a pulley and weights are hung on the end. The motion trackers can be seen in the fibula and talus.

Figure 1(B). Test setup showing the cable, pulley, and weights that create the body weight axial compressive force on the foot and ankle.

Data Analysis

Stiffness was calculated from the slope of the torque/rotation curve from 5° to 15° rotation of the second cycle (Figure 2). The peak torque at 20° ankle inversion was reported. Intra-articular tibiotalar peak pressure (MPa), mean contact area (mm^2), and center of force (mm) were recorded at 15 Hz using the pressure measurement system.

The peak pressure frame of the second of three cycles of inversion was used for analysis of contact area, peak pressure, and center of force (COF) because this is when the inversion motion had the smoothest arc. The COF was reported as a single, static point in the peak pressure frame. The 3D motion capture camera system was used to assess the following:
(1) the angle of inversion of the talus relative to thefibula;

(2) the angle of inversion of the calcaneus relative to the fibula and;
(3) the medial displacement of the calcaneus relative to the fibula.

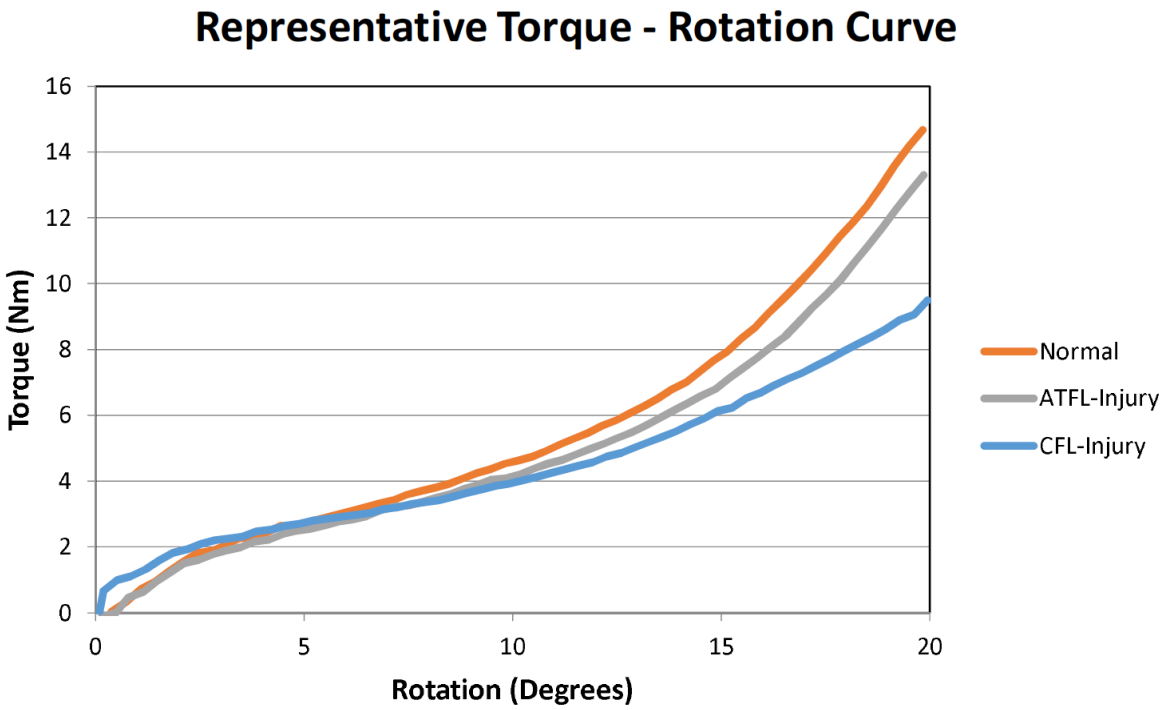

Figure 2. Typical Torque-Rotation curve of the same specimen in the Normal, ATFL-injury, and CFL-injury state.

Statistical Analysis

All analyses were performed using SAS 9.4. (SAS Institute Inc.Cary, NC, USA). Student's t-test with Bonferroni correction was used to compare the differences in COF (mm) across the three conditions; a p-value of < 0.017 was regarded as statistically significant. Linear mixed model regression analyses were used to compare ankle peak torque (N.m) and stiffness (N.m/deg) across the three conditions. Linear mixed model regression analyses were also used to determine significance for peak pressure (MPa), contact area (mm^2), the inversion angles (in degrees) of the talus and calcaneus relative to the fibula, as well as the medial displacement (in mm) of the calcaneus relative to the fibula across the three conditions; a p-value < 0.05 was regarded as statistically significant.

RESULTS

Stiffness and Peak Torque

Mean stiffness and peak torque values for the three conditions can be found in Table 1.
When compared to the intact condition, the difference in mean stiffness for the CFL-injury condition was significant ($p = 0.0002$). Similarly, the mean difference in stiffness between the ATFL-injury and CFL-injury conditions was also significant ($p = 0.0075$). There was no significant difference in mean stiffness when comparing the ATFL-injury and intact conditions
($p = 0.2254$).
When comparing the CFL-injury and intact conditions, the mean difference in peak torque was significant ($p < 0.0001$). When comparing the CFL-injury and ATFL-injury conditions, the mean difference in peak torque was also significant ($p = 0.0012$).
However, there was no significant difference in mean peak torque when comparing the ATFL injury and the intact condition ($p = 0.3371$).

Condition	Mean (SD)	95% Confidence Interval	
		Lower Bound	Upper Bound
Stiffness (N·m/deg)			
Normal	0.67 (0.38)	0.49	0.85
ATFL-injury	0.61 (0.35)	0.45	0.78
CFL-injury	0.49 (0.33)	0.34	0.64
Peak Torque (N·m)			
Normal	16.03 (8.37)	11.99	20.06
ATFL-injury	15.46 (7.82)	11.80	19.11
CFL-injury	12.22 (7.57)	8.68	15.77

Table 1. Stiffness (N·m/deg) and Peak Torque (N·m)

Peak Pressure, Contact Area, and Center of Force (COF)

Mean peak pressure and contact area values for the three conditions can be found in Table 2.
When comparing the CFL-injury and the intact condition, the mean difference in peak pressure was significant ($p = 0.0003$). Similarly, when comparing the CFL-

injury and ATFL-injury conditions, the mean difference in peak pressure was also significant (p= 0.002).

However, there was no significant difference in mean peak pressure when comparing the ATFL-injury and intact conditions (p = 0.4848). When comparing the CFL-injury and intact conditions, there was a significant difference in mean contact area (p= 0.0084). When comparing the CFL-injury and ATFL-injury conditions, the results also showed that there was a significant difference (p = 0.0037). However, there was no significant difference in mean contact area when comparing the ATFL-injury and intact conditions (p= 0.7587).

Condition	Mean (SD)	95% Confidence Interval	
		Lower Bound	Upper Bound
Peak Pressure (MPa)			
Normal	19.56 (13.13)	13.41	25.70
ATFL-injury	18.89 (12.94)	12.83	24.94
CFL-injury	15.72 (9.76)	11.15	20.28
Contact Area (mm^2)			
Normal	137.58 (49.12)	114.59	160.57
ATFL-injury	135.27 (44.76)	114.32	156.22
CFL-injury	158.31 (65.80)	127.52	189.11

Table 2. Peak Pressure (MPa) and Contact Area (mm^2)

Center of Force (COF)

Representative COF images can be found in Figure 3. During the ATFL-injury condition, the COF moved 0.76 mm medially, relative to the intact condition (p = 0.008). While there was a net movement of 0.99 mm medially from the intact condition to the CFL-injury condition, this was not significant (p = 0.059). During the ATFL-injury condition, the COF moved 0.32 mm anterior relative to the intact condition (p = 0.773).
During the CFL-injury condition, the COF moved 1.03 mm posterior, relative to the ATFL-injury condition, resulting in a net movement of 0.71 mm, posterior from the intact condition to the CFL-injury condition (p =0.009).

Figure 3. Representation of COF during Inversion

Motion Capture Data

All mean values from the motion capture data can be found in Table 3.

Talus inversion: When comparing the CFL-injury condition to the intact condition, the mean difference in the inversion angle was significant ($p < 0.0001$). Additionally, the mean difference in the inversion angle was also significant when comparing the CFL-injury and ATFL-injury conditions ($p = 0.0021$). There was no significant difference when comparing the intact and ATFL-injury conditions ($p = 0.1215$).

Calcaneus inversion: When comparing the CFL-injury and intact condition, the mean difference in the inversion angle was found to be significant ($p < 0.0001$). The mean difference in the inversion angle was also significant when comparing the CFL-injury and ATFL-injury conditions ($p = 0.0016$). However, the mean difference in inversion angle when comparing the intact and ATFL-injury conditions was not significant ($p = 0.2887$).

Medial displacement of calcaneus: Additionally, when comparing the mean medial displacement between intact and ATFL-injury conditions, as well as the ATFL-injury and CFL-injury conditions, these differences were not found to be significant either ($p = 0.2721$ and $p = 0.5639$, respectively).

Condition	Mean (SD)	95% Confidence Interval	
		Lower Bound	Upper Bound
Talus Inversion Angle (°)			
Normal	4.39 (4.73)	1.65	7.12
ATFL-injury	4.89 (4.98)	2.02	7.77
CFL-injury	5.98 (5.52)	2.79	9.16
Calcaneus Inversion Angle (°)			
Normal	13.12 (2.87)	11.46	14.78
ATFL-injury	13.70 (3.33)	11.77	15.62
CFL-injury	15.58 (4.33)	13.08	18.08
Medial Displacement of Calcaneus (mm)			
Normal	8.22 (4.93)	5.91	10.52
ATFL-injury	9.36 (8.19)	5.53	13.19
CFL-injury	9.96 (8.47)	6.00	13.93

Table 3. *Motion Capture Measurements*

DISCUSSION

The goal of this study was to determine the role of the ATFL and CFL in inversion ankle stability. These data support the hypotheses that the CFL plays a significant role in ankle joint stability during load-bearing inversion conditions.

Stiffness and peak torque decreased significantly only after sectioning of both ATFL and CFL. Peak pressures in the tibiotalar joint decreased significantly only following CFL release, and mean tibiotalar contact area significantly increased only following CFL release.

Motion capture data showed a significant increase in inversion angle of both the calcaneus and talus after sectioning the CFL but not after sectioning the ATFL. While the data did not show significant increases in the calcaneus medial displacement in both the ATFL-injury and CFL-injury condition, there was a trend.

The ATFL and CFL are considered the primary lateral ankle stabilizers. The current study examined their role in inversion only. In another study examining the role of the ATFL and CFL on ankle stability, Ziai et al. examined internal rotation in a cadaver model, in which they measured the torque necessary to internally rotate the tibia 30° intact and with both the ATFL and CFL sectioned.[32] They found that sectioning both the ATFL and CFL significantly reduced the torque necessary to achieve 30° degrees of internal tibia rotation. These studies

demonstrate the important role that both the ATFL and CFL play on ankle stability in both inversion and internal rotation.

The individual role that the ankle joint and subtalar joint play in the stiffness and peak torque measurements made in the current study may explain why there were no significant differences in stiffness or peak torque between the Normal and the ATFL-injury while there were significant differences between the Normal and CFL-injury. The ankle joint primarily allows for plantar/dorsiflexion and the subtalar joint primarily allows for inversion/eversion.
When the ATFL was sectioned, the lateral and medial malleolus maintained most of the inversion stiffness and peak torque that the ankle joint contributes to overall stiffness and peak torque. When the ATFL was sectioned, the inversion angle only increased 0.50° for the talus and 0.58° for the calcaneus, which did not result in an overall significant change in stiffness or peak torque. When the CFL was sectioned, the inversion angle increased 1.59° in the talus and 2.46° in the calcaneus. This resulted in a significant decrease in the stiffness and peak torque. These results are similar to the results of Bahr et al.[3] They tested the foot and ankle with a 375 N compressive joint load and 3.4 N m inversion torque. After sectioning the ATFL, the tibiocalcaneal motion increased approximately 1° and the tibiotalar motion increased approximately 2°. After sectioning both the ATFL and CFL, the tibiocalcaneal motion increased approximately 8° and the tibiotalar motion increased approximately 15°. In addition, the non-significant changes in the ATFL-injury may be due to the differences in stiffness of the ATFL and CFL. Attarian et al. showed in a typical load deflection curve that the CFL is stiffer than the ATFL, approximately 40 N m compared to 25 N m, respectively.[2] Sectioning the less stiff ATFL first resulted in smaller changes in stiffness and peak torque than when the more stiff CFL was sectioned.

The current study can be compared to other studies in the literature that also reported inversion stiffness results from tests with the foot in 20° of plantarflexion and 15° of inversion.[7, 14] For example, Giza et al. tested the ankles after sectioning the ATFL and CFL and repairing them, while Brown et al. tested the ankles after sectioning and repairing only the ATFL.[7, 14]

However, neither study tested the intact ankle; they only tested the repaired ankles that showed stiffness that is less than the stiffness found in the current study. In addition, neither study conducted testing with load-bearing inversion. Giza et al. showed a stiffness of the repaired ankle ranging from 0.4 N·m/deg to

0.45 N·m/deg, while Brown et al. reported a stiffness of 0.315 N·m/deg and 0.417 N·m/deg.[7, 14]

However, the current study reports the stiffness of the ATFL deficient ankle being 0.615 N·m/deg and the stiffness of the ATFL/CFL deficient ankle being 0.49 N·m/deg. The reported stiffness in the current study is larger than that found in the two other studies because a weight-bearing force was applied across the joint during testing, simulating weight-bearing inversion conditions.

This force, intended to simulate the typical injury mechanism of weight-bearing inversion, increases the friction across the joint resulting in higher stiffness. The alteration in the location of COF was an important finding in this study. It is known that repeated ankle injuries can increase risk of cartilage damage with further injury. While incompetent ligaments can certainly increase the risk of more severe injury, alteration of the location of forces in the tibiotalar joint during load-bearing inversion suggest that risk can be increased even in sub-injury conditions.

Our data suggest a movement of the COF medially toward the medial shoulder of the talar dome, which has been reported as the most common location of osteochondral lesions of the talus.[11]
Since talar OCDs are commonly identified in patients with ankle injuries, the COF may play a role in the etiology or exacerbation of these lesions. The study by Prisk et al. measured the COF during ankle inversion in the intact and CFL-injury state.[27] They found the COF to move medially and anteriorly while the current study found the COF to shift medially and posteriorly. This difference may be due to the different loading conditions. Prisk et al. used a 200 N axial compressive force and 4.5 N m of inversion.

The current study applied a compressive axial load of donor body weight (ranging from 400 N to 1112 N) and inversion to 20°, which was 16.0 N m and 12.2 N m, for intact and CFL-injury, respectively.

There are several limitations to this study. With the use of cadavers, the complex muscle forces and ground reaction forces that cross the ankle joint in vivo were not simulated. Additionally, we were only able to test in one configuration, 20° plantarflexion 15° internal rotation; however, this has been shown to be the most common position of the ankle during lateral ankle injuries.[13] Furthermore, only the ATFL and CFL were examined in this study. The posterior talofibular ligament (PTFL) also contributes to lateral ankle instability

but was not examined in this study because it is less commonly injured in isolated ankle sprains. In addition, we did not incorporate injury to the interosseous ligament or other ligaments that stabilize the subtalar joint (that are often injured in high Grade sprains) in order to isolate the impact of CFL injury on the ankle joint only. In addition, in order to gain access to the tibiotalar joint to insert the pressure sensors, the extensor digitorum longus, tibialis anterior, extensor hallucis longus, and Achilles tendons were sectioned.

However, these structures are not considered lateral ankle stabilizers and should not have influenced the results. The accuracy of Tekscan sensor has been shown to decrease with repeated measures and may have affected the results. Jansson et al. showed that a Tekscan sensor calibrated in a dry environment and tested in either a humid or wet environment recorded 100% or 95% of the initial load at 0.75 hours.[18] Each specimen in the current study was completed within 0.25 hours, from start to finish.

CONCLUSION

Evolving lateral ankle instability surgical techniques focus on the importance of restoring the ATFL. However, the results of this biomechanical study under weight-bearing conditions, suggest that the CFL plays an important role in the stability of both the ankle and subtalar joints, and in tibiotalar contact mechanics.

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The CFL Fails Before the ATFL Immediately After Combined Ligament Repair in a Biomechanical Cadaveric Model

Journal of Knee Surgery, Sports Traumatology and Arthroscopy (KSSTA)
(accepted for publication, July 2019)

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ABSTRACT

Purpose:
The purpose of this study was to assess the impact on ankle stability of repairing the ATFL alone compared to repairing both the ATFL and CFL, in a biomechanical cadaver model.

Methods:
Ten matched pairs of intact, fresh frozen human cadaver ankles (normal) were mounted to a test machine in 20° plantar flexion and 15° of internal rotation. Each ankle was loaded to body weight and then tested from 0 to 20° of inversion. The data recorded were torque at 20° and stiffness, peak pressure and contact area in the ankle joint using a Tekscan sensor, rotation of the talus and calcaneus, and translation of the calcaneus using a three-dimensional motion capture system. Ankles then underwent sectioning of the ATFL and CFL (injured), retested, then randomly assigned to ATFL only Broström repair or combined ATFL and CFL repair. Testing was repeated after repair then loaded in inversion to failure (LTF). The mode of failure was recorded.

Results:
The stiffness of the ankle was not significantly increased compared to the injured condition by repairing the ATFL-only (P=0.67) or the ATFL/CFL (P=0.08). The calcaneus had significantly more rotation than the injured condition in the ATFL-only repair (P=0.04) but not in the ATFL/CFL repair (P=0.55). The ATFL failed at 40.3% more torque than the CFL, at 17.4±7.0 Nm and 12.4±4.1 Nm, respectively, and 62.0% more rotation, at 43.9±5.6° and 27.1±6.8°, respectively.

Conclusions:
This study found a greater increase in stiffness following combined ATFL/CFL repair compared to ATFL-only repair, although this did not reach statistical significance. The CFL fails before the ATFL, potentially indicating its vulnerability immediately following repair.

Clinical Relevance:
Restoring the CFL likely plays a relevant role in lateral ligament repair, however sufficient time for ligament healing should be allowed before rehabilitation stresses are applied.

INTRODUCTION

Lateral ankle sprains (LAS) are among the most common musculoskeletal injuries in both athletes and non-athletes. It is estimated that there are 30,000 ankle sprains per day in the United States and account for 7-10% of the visits to the emergency room.[3, 7, 9] LAS is caused by a forceful ankle inversion beyond its normal limit, injuring the lateral ligaments that stabilize the ankle. The anterior talofibular ligament (ATFL) is almost always injured and the calcaneofibular ligament (CFL) is injured 50% to 75% of the time.[5] The posterior talofibular ligament (PTFL) also stabilizes the lateral ankle but it is rarely injured.[5] Injuries to these ligaments can be a stretching and slight tear (Grade I), partially torn (Grade II), or completely torn (Grade III). Symptoms of a LAS may include pain, swelling, tenderness to the touch, loss of range of motion (ROM), ankle instability, and difficulty in full weight bearing.

Conservative, non-operative management of acute ankle sprains is appropriate for the majority of ankle sprains. Non-operative treatment may involve bracing, resting, icing, non-steroidal anti-inflammatory drugs, proprioception training, and physical therapy. However, it is estimated that 20 to 40% of severe LAS will lead to chronic ankle instability (CAI).[18] CAI is characterized by one significant LAS, the ankle "giving way", recurrent LAS, and reduced ankle function. If 3 to 6 months of non-surgical treatment is unsuccessful, over half of orthopaedic surgeons surveyed with experience in treating patients with CAI recommend considering surgery .[17] Another third of surgeons surveyed recommend non-surgical treatment for 6 to12 months before considering surgery.[17]

When surgery is recommended for CAI in which both the ATFL and CFL are injured, there is disagreement about the necessity of repairing the CFL. In the survey by Michels et al., 60% of respondents (almost) always repair the CFL, 13.3% (almost) never repair the CFL, 20.0% say it depends on subtalar instability and 10.0% say it depends on CFL quality.[17] In addition, there are studies in the literature that both support repairing only the ATFL and repairing both the ATFL and CFL. Okudu reported on a series of 27 patients with CAI, where 11 patients had only an injured ATFL and 16 patients had both an injured ATFL and CFL.[20] They repaired only the ATFL in both groups and showed excellent functional results in 26 of 27 ankles and good results in one ankle, and concluded that it is not necessary to reconstruct the CFL along with the ATFL.
In a biomechanics cadaver study, Lee et al. showed that repairing the ATFL alone with a modified Broström procedure can provide as much initial stability

as repairing both the ATFL and CFL.[15] However, Kjaersgaard-Andersen et al. showed the CFL has a significant effect on stabilizing the talocalcaneal joint in a cadaver study.[13] Chrisma et. al. reported that it is essential to repair both the ATFL and CFL.[6]

Despite the common occurrence of lateral ankle sprains involving both the ATFL and CFL, the precise impact of repairing the CFL in ankle instability remains unclear. Using a biomechanical cadaver model, we aimed to assess the impact of repairing the ATFL alone compared to repairing both the ATFL and CFL. We hypothesized that repairing the CFL will substantially augment ankle and subtalar joint stability during weight-bearing ankle inversion compared to ATFL repair alone.

METHODS

Specimen Preparation

A power analysis determined that ten matched pairs (20 total) of fresh frozen human cadaver ankles were required to show a 20% difference between repairs with a level of significance of 0.05 and 80% power.[8] Ten matched pairs of mid-tibia to toe tip specimens were obtained from a local tissue bank (Science Care, Inc., Aurora, CO, USA).
The use of these specimens for biomechanical testing met all the requirements of the Institutional Review Board (IRB) at the senior author's institution. There were 5 male donors, average age 51.4 years, range 38-60, and 5 female donors, average age 53.8 years, range 32-64.
Previous studies have established the use of fresh frozen specimens compared to specimens not frozen, as there was minimal effect on the gross biomechanical properties of the ligaments and other connective tissues due to freezing.[21, 22] All specimens were evaluated visually and radiographically for signs of gross deformity, previous operation, fracture, and arthritis. Specimens were wrapped in gauze soaked in 0.9% sodium chloride solution and placed in a -25°C freezer for storage. The specimens were thawed at room temperature on the day they were prepared and tested. All soft tissue was dissected 4" from the proximal end of the tibia. Next, the exposed proximal fibula was rigidly fixed to the tibia with a 4.5 mm cortex screw. The proximal 3" of the tibia/fibula was potted in a 3-inch diameter aluminum tube with SmoothCast 321 (Smooth-On, Inc., Easton, PA USA) that allowed the specimen to be internally rotated. The plantar surface was secured in a SmoothCast 321 bed with one additional screw in the calcaneus for fixation.

To measure the contact area and pressure in the ankle joint, a thin film Tekscan pressure mapping sensor Model 5033 was inserted into the ankle joint (Tekscan Inc., South Boston, MA USA). The pressure sensing area of the sensor measured 26.7mm wide and 38.3mm high. The sensor was calibrated with a 10 point 10-point loading calibration procedure. To insert the sensor, the ankle joint was exposed both anteriorly and posteriorly by removing all surrounding soft tissue including the joint capsule. The sensor was then placed into the ankle joint posteriorly. Pressure sensor data was recorded on a PC equipped with I-Scan software at 15 frames per second. From the pressure sensor data the mean contact area and peak pressure were recorded.

To measure the motion of the talus and calcaneus relative to the fibula, a 2-camera, 3-D motion capture system was used (Innovision Systems Inc, Columbiaville, MI USA). A 3-D calibration frame was in the field of view of both cameras during testing. Passive reflective markers were rigidly fixed to the fibula, talus, and calcaneus. Synchronized video was captured at 1530 frames per second throughout testing (Figure 1). From the motion capture data, the rotation of the talus and calcaneus as well as the translation of the calcaneus were reported.

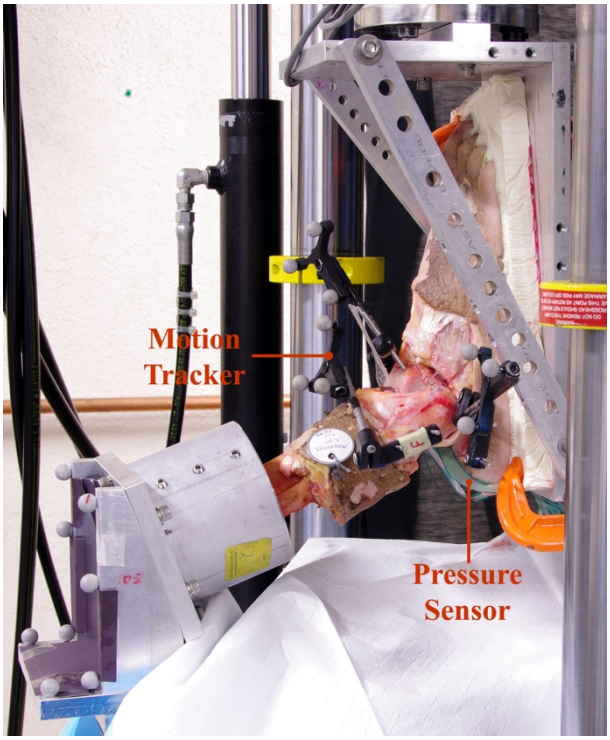

Figure 1. Testing setup of the cadaveric specimen and synchronized video capturing

Biomechanical Testing

Biomechanical testing was performed on an Instron Model 1321 servo-hydraulic test machine with 8500 controllers (Instron Corp., Norwood, MA USA). Each intact specimen (Normal group) was mounted horizontally onto the testing apparatus in 20° of plantar flexion and 15° of internal rotation with the center of rotation of the tibiotalar joint aligned with the rotation of axis of the Instron actuator.[4] This alignment was used to align the ankle in a position that it is susceptible to a lateral ankle sprain.[10-12] Specimens were axially loaded to body weight using hanging weights attached to the test fixture to put a compression force across the ankle joint. Each ankle was preconditioned for 10 cycles from 0 to 10° of inversion at 0.25 Hz.[19] Each ankle was tested from 0 to 20° of inversion at 0.125 Hz for three cycles.
The ATFL and CFL were then sectioned (Injured group), and inversion testing was repeated. The torque and rotation of the Instron was recorded at 30 Hz with a PC equipped with an analog to digital data acquisition board and data acquisition software.
The stiffness was measured as the slope of the torque/rotation curve. The torque at 20 degrees inversion was also reported.

Following sectioning of the ATFL and CFL, matching ankles were assigned to either an ATFL only repair group or an ATFL and CFL repair group using a coin flip generator. As a result, 10 ankles were assigned to the ATFL only (ATFL-only group) repair and 10 were assigned to the ATFL and CFL repair (ATFL and CFL repair group). The ankles were removed from the test setup for repair.

Repair technique

ATFL Repair technique
In all specimens, the ATFL was repaired using a single all-soft suture anchor (1.4 mm juggerknot™, Zimmer-Biomet). A 1.4 mm drill was used to create a pilot hole, and the anchor was advanced into the trill hole using a mallet to the appropriate depth. Using the #1 Maxbraid™ (Zimmer-Biomet) sutures attached to the juggerknot anchor, the sectioned ligament was repaired to the proximal limp and fibular periosteum in a pants over vest fashion. The repair was supplemented by two additional #1 Maxbraid sutures also in a pants-over-vest technique. All repairs were performed with the ankle in neutral and the hindfoot in eversion.

CFL repair technique

For specimens in the CFL group, the sectioned CFL ligament was repaired with a second all-soft suture anchor (juggerknot™, Zimmer-Biomet) placed in the distal fibula. The sectioned ligament was repaired in a pants-over-vest patter to the proximal ligament and periosteum. The repair was supplemented with an additional #1 Maxbraid suture also in a pants-over-vest technique.
Following repair, the ankles were once again placed in the test setup and inversion testing was repeated. After inversion testing of the repaired ankles, the ankles underwent a load to failure test. In this test, the ankles underwent inversion at 5° per second until both the CFL (for the ankles that underwent double repair) and the ATFL failed. The torque and inversion angle at which failure occurred were recorded. The video was reviewed to determine the mode of failure.

Statistical Analysis

All statistical analyses were conducted using SAS 9.4 (SAS Institute Inc., Cary, NC USA). For the ATFL only repair group and the ATFL and CFL repair group, paired t-test was used to analyze the differences in the following outcomes:
(1) Torque at 20° Inversion (N·m);
(2) Ankle Stiffness (N·m/deg);
3) the medial displacement (in mm) of the calcaneus relative to the fibula;
(4) the inversion angles (in degrees) of the calcaneus and talus, relative to the fibula;
(5) peak pressure (MPa); and contact area (mm^2).

Paired t-test was also used to compare the differences in load to failure and rotation for the two repair groups. Linear mixed model regression analyses were used to analyze pairwise comparisons for the aforementioned outcomes across the following conditions:
(1) Normal;
(2) Injured;
(3) Repaired.
A p-value of < 0.05 was regarded as statistically significant.

RESULTS

Normal vs. Injured Condition

For both repair groups, there were significant differences between the normal and injured conditions. For torque at 20° inversion and stiffness, there were significant differences between the two conditions for both groups (Table 1).

With regards to the medial displacement of the calcaneus relative to the fibula, as well as the inversion angles of the calcaneus and talus (also relative to the fibula), only the ATFL and CFL repair group demonstrated significant differences between the normal and injured conditions (Table 2). For peak pressure, only the ATFL only repair group demonstrated a significant difference between the normal and injured condition (p-value = 0.017). There were no significant differences in contact area between the normal and injured conditions for either repair group (Table 3).

Normal vs. Repaired Condition

For both repair groups, there were significant differences between the normal and repaired conditions. Similar to the normal and injured conditions, there were significant differences in both torque at 20° inversion and stiffness for both repair groups as well (Table 1). Regarding the motion capture measurements, only the ATFL and CFL repair group showed statistically significant differences between the normal and repaired conditions; however, only the mean differences in the calcaneus and talus inversion angles were statistically significant (Table 2).

For peak pressure, only the ATFL only repair group demonstrated a statistically significant difference (p-value = 0.0091). There were no significant differences in contact area when comparing the normal and repaired conditions for either repair group (Table 3).

Injured vs. Repaired Condition

For both repair groups, there were limited significant differences between the injured and repaired conditions. There were no significant differences in both torque at 20° inversion and stiffness for either repair group. (Table 1). For the motion capture data, only the mean difference in the calcaneus inversion angle for the ATFL only repair group was significantly different (p-value = 0.0365); the rest of the mean differences were not statistically significant (Table 2).

Table 1. Torque at 20° inversion (N·m), Stiffness (N·m/deg), and Load to Failure data

Comparison	Mean Difference	95% Confidence Interval		p-value
		Lower Bound	Upper Bound	
Torque at 20° Inversion (N·m)				
Normal vs. Injured Condition				
ATFL Only Repair	3.31	1.48	5.14	0.0015**
ATFL and CFL Repair	2.68	0.76	4.60	0.0095**
Normal vs. Repaired Condition				
ATFL Only Repair	3.14	1.27	5.00	0.0027**
ATFL and CFL Repair	1.93	-0.03	3.89	0.053
Injured vs. Repaired Condition				
ATFL Only Repair	-0.18	-1.03	0.67	0.6632
ATFL and CFL Repair	-0.67	-1.52	0.18	0.1140
ATFL Only Repair vs. ATFL and CFL Repair	-1.80	-5.78	2.18	0.3519
Stiffness (N·m/deg)				
Normal vs. Injured Condition				
ATFL Only Repair	-0.11	-0.20	-0.01	0.0267*
ATFL and CFL Repair	-0.14	-0.23	-0.04	0.0068**
Normal vs. Repaired Condition				
ATFL Only Repair	0.10	0.01	0.18	0.029*
ATFL and CFL Repair	0.09	0.0006	0.17	0.0485*
Injured vs. Repaired Condition				
ATFL Only Repair	-0.01	-0.07	-0.05	0.6698
ATFL and CFL Repair	-0.05	-0.11	0.0008	0.0849
ATFL Only Repair vs. ATFL and CFL Repair	-0.07	-0.27	0.15	0.5163
Failure Torque				
ATFL Only Repair vs. ATFL and CFL Repair	5.02	1.81	8.24	0.0030**
Degrees of Rotation at Failure				
ATFL Only Repair vs. ATFL and CFL Repair	15.07	12.08	18.06	< 0.0001**

*p-value < 0.05
**p-value < 0.01

Table 2. Motion Capture Measurements

Comparison	Mean Difference	95% Confidence Interval		p-value
		Lower Bound	Upper Bound	
Medial Displacement of Calcaneus (mm)				
Normal vs. Injured Condition				
ATFL Only Repair	0.57	-0.91	2.04	0.4255
ATFL and CFL Repair	-1.68	-3.15	-0.20	0.0282*
Normal vs. Repaired Condition				
ATFL Only Repair	1.03	-0.99	3.06	0.2952
ATFL and CFL Repair	-1.91	-3.94	0.12	0.0628
Injured vs. Repaired Condition				
ATFL Only Repair	0.47	-1.07	2.01	0.5308
ATFL and CFL Repair	-0.23	-1.77	1.31	0.7534
ATFL Only Repair vs. ATFL and CFL Repair	-2.18	-8.52	4.12	0.4701
Calcaneus Inversion Angle (°)				
Normal vs. Injured Condition				
ATFL Only Repair	-1.38	-3.26	0.49	0.1324
ATFL and CFL Repair	-2.93	-4.95	-0.90	0.0087**
Normal vs. Repaired Condition				
ATFL Only Repair	-0.58	-2.58	1.42	0.5356
ATFL and CFL Repair	-2.70	-4.85	-0.54	0.0187*
Injured vs. Repaired Condition				
ATFL Only Repair	0.80	0.06	1.55	0.0365*
ATFL and CFL Repair	0.23	-0.58	1.03	0.5465
ATFL Only Repair vs. ATFL and CFL Repair	-2.73	-7.38	1.93	0.2236
Talus Inversion Angle (°)				
Normal vs. Injured Condition				
ATFL Only Repair	-0.82	-1.78	0.14	0.0875
ATFL and CFL Repair	-2.02	-3.06	-0.98	0.0013**
Normal vs. Repaired Condition				
ATFL Only Repair	-0.95	-1.92	-0.03	0.0554
ATFL and CFL Repair	-1.43	-2.47	-0.38	0.0123*
Injured vs. Repaired Condition				
ATFL Only Repair	-0.1271	-0.95	0.70	0.7413
ATFL and CFL Repair	0.60	-0.30	1.49	0.1693
ATFL Only Repair vs. ATFL and CFL Repair	2.83	-2.76	8.42	0.2866

**p-value < 0.01

For contact area, only the ATFL and CFL repair group showed a statistically significant difference (p-value = 0.0208). There was no significant differences in peak pressure when comparing the injured and repaired conditions for either repair group (Table 3).

Repaired Condition – ATFL Only Repair vs. ATFL and CFL Repair

When comparing the repair groups within the repaired condition, there were no significant differences in either torque at 20° inversion or stiffness. However, there were statistically significant differences in both the failure torque and degrees of rotation at failure between the two groups (Table 1). The ATFL failed at 40.3% more torque than the CFL, at 17.4±7.0Nm and 12.4±4.1Nm, respectively. The ATFL failed at 62.0% more rotation than the CFL, at 43.9±5.6° and 27.1±6.8°, respectively. There were no statistically significant differences in the medial displacement of the calcaneus relative to the fibula, the calcaneus and talus inversion angles (also relative to the fibula) (Table 2), peak pressure or contact area (Table 3).

Table 3. Peak Pressure (MPa) and Contact Area (mm^2)

Comparison	Mean Difference	95% Confidence Interval		p-value
		Lower Bound	Upper Bound	
Peak Pressure (MPa)				
Normal vs. Injured Condition				
ATFL Only Repair	5.94	1.25	10.62	0.0170*
ATFL and CFL Repair	2.05	-2.34	6.43	0.3315
Normal vs. Repaired Condition				
ATFL Only Repair	7.74	2.28	13.20	0.0091**
ATFL and CFL Repair	1.53	-3.58	6.64	0.5288
Injured vs. Repaired Condition				
ATFL Only Repair	1.80	-2.19	5.79	0.3477
ATFL and CFL Repair	-0.52	-4.25	3.21	0.7692
ATFL Only Repair vs. ATFL and CFL Repair	8.42	-0.47	17.31	0.0606
Contact Area (mm^2)				
Normal vs. Injured Condition				
ATFL Only Repair	-20.57	-58.56	17.41	0.2630
ATFL and CFL Repair	-29.68	-65.21	5.85	0.0943
Normal vs. Repaired Condition				
ATFL Only Repair	-2.87	-32.75	27.01	0.8388
ATFL and CFL Repair	4.98	-22.97	32.93	0.7067
Injured vs. Repaired Condition				
ATFL Only Repair	17.7	-12.75	48.15	0.2313
ATFL and CFL Repair	34.66	6.18	63.14	0.0208*
ATFL Only Repair vs. ATFL and CFL Repair	-13.12	-60.28	34.04	0.5544

*p-value < 0.05
**p-value < 0.01

Mode of failure

During load to failure testing, all the specimens in the ATFL only repair group failed at the suture/tissue interface. In the ATFL and CFL repair group, the knot failed in 3 of 10 CFLs and at the suture/soft tissue interface in the remaining 7 specimens. All 20 of the ATFL repairs failed at the suture/soft tissue interface.

DISCUSSION

Lateral ankle sprains are a common injury in both the athletes and non-athletes. In more severe ankle sprains, both the ATFL and CFL are ruptured but there is no consensus if the CFL needs to be repaired. The purpose of this study was to compare the ankle biomechanics of repairing only the ATFL to repairing both the ATFL and CFL in a cadaver model.
The results of this study show that immediately after repairing only the ATFL or both the ATFL and CFL, the ankle biomechanics are not significantly improved compared to the injured ankle. This result emphasizes the importance of allowing adequate time for the healing of the ATFL and CFL after surgery. Because previous studies have shown that the CFL is a significant contributor to the lateral ankle stability, the CFL should also be repaired.[2]

To our knowledge, this is the first study to compare the ankle biomechanics in a weight bearing cadaver model of the intact ankle, ATFL and CFL injured ankle, and ATFL only or ATFL and CFL repaired ankle. The data showed that repairing only the ATFL or both the ATFL and CFL did not significantly increase the ankle joint biomechanics when compared to the injured state. This result may be due to the suture material being less stiff than the intact ligament or the lack of rigid fixation at the suture/tissue interface. In the load to failure tests, 18/20 specimens failed at the suture/tissue interface.

The study also showed that the injured ankle was less stable than the normal ankle, but these differences were not significant for all factors. Similarly, Kovaleski et al. showed that the ATFL and CFL injured ankle resulted in a significant increase in inversion angle and decrease in inversion stiffness and significantly increased anterior displacement in a non-weight bearing cadaver model.[14] Despite showing significant differences in anterior displacement, the end range anterior stiffness did not significantly change.
There are other studies in the literature that compare the stability of the intact ankle to the ATFL and CFL injured ankle, and the repaired ankle. Bahr et al. tested cadaver ankles by applying a 3.4N-m inversion torque and measured the

angle of inversion of the talus and calcaneus in the intact, ATFL only deficient, ATFL and CFL deficient, and three different ATFL and CFL repair techniques: Bröstrom repair, Watson-Jones reconstruction, and a new anatomic reconstruction.[2]
Compared to the intact state, their results showed increased inversion of the talus and calcaneus in the ATFL only deficient state. The calcaneus inversion increased approximately 1° and the talus inversion increased approximately 2°. After the CFL was sectioned, the calcaneus inversion increased approximately 7° and the talus inversion increased approximately 13°.

The three repair techniques showed a decrease in inversion of the talus and calcaneus compared to the ATFL and CFL deficient ankle but not to the level of the ATFL only deficient or normal ankle with the exception of the Watson-Jones reconstruction.[2] The Watson-Jones reconstruction resulted in the talar inversion being similar to the normal condition. The current study conducted a similar inversion test but controlled the amount of ankle inversion and reported the amount of torque required to achieve 20° of inversion. The current study showed significantly more inversion of the talus and calcaneus in the ATFL and CFL injured state compared to the normal state. The ATFL only or the ATFL and CFL repair did not restore the inversion angles back to the normal state.
In another cadaver study, Lee et al looked at the talar inversion angle of the intact, ATFL and CFL injured ankle, the ATFL only repaired, and the ATFL and CFL repaired ankle, using a modified Broström repair for both groups.[15] However, while they only did statistical comparisons between the two repair groups, they reported the talar inversion angle values of all conditions. The normal ankle talus inversion ankle averaged 3.2°, the injured group was 14.0°, and the repaired groups were 1.8° for the ATFL and CFL repair and 1.5° for the ATFL only repair.
Their results showed the repair groups having less talar inversion than the normal group. This difference from the current study could be due to the different repair methods used or the different testing methods used. Lee et al. applied a 150 N varus stress force using the Telos device while the current study applied 20° of inversion under a compressive force of body weight, intended to simulate inversion injury.
The load to failure data appears to contradict what is seen clinically. Clinically, the ATFL is the most injured ligament in the lateral ankle and the CFL is also involved 50 to 70% of the time.[10] This clinical finding implies the ATFL is weaker than the CFL. In the current study, the CFL failed at a significantly lower torque and lower inversion angle compared to the ATFL, implying the CFL is weaker than the ATFL.

In a direct biomechanical comparison of the strengths of the ATFL and CFL, the CFL failed at an average force of 345.7 ± 55.2 N and the ATFL failed at 138.9 ± 23.5N.[1] The CFL being the weak link in the current study could be primarily due to two reasons. First, the CFL is thinner than the ATFL, which would result in the CFL failing first with 18 of 20 failures occurring at the suture/tissue interface. Yildiz et al. reported that the width of the ATFL is 11.07 ± 5.63mm and the width of the CFL is 5.44 ± 2.34mm.[23] Second, the inversion method of testing puts more force on the CFL than the ATFL, which could be why the CFL failed first. The CFL attaches to the calcaneus, which inverted more than the talus, which the ATFL attaches to.

This is supported by a biomechanics cadaver study by Bahr et al.[2] They put force transducers on the intact ATFL and CFL and measured the forces on the ligaments during 15° of inversion with the foot in neutral flexion. They measured approximately 40 N of force on the CFL and approximately 20 N of force on the ATFL.
The mode of failure in the current study and other studies emphasize the importance of allowing adequate time for the repair to heal. In the current study, 18 of 20 failures occurred at the suture/soft tissue interface.

In a study by Li et. al., only 1 of 12 specimens failed at the suture/tissue interface.[16] They had 5 of 6 fail by anchor pullout and 6 of 6 fail at the bony bridge in the transosseous tunnel group. Brown et al. showed more similar results to the current study.[4] In their anchor group, 8 of 9 specimens failed at the suture/soft tissue interface. In all three of these studies, none of the ligaments failed; they all failed at the ligament/suture or anchor/bone interface. Allowing adequate time to heal following surgery would strengthen the ligament/bone interface and decrease the chance of a suture/ligament or anchor/bone failure.

There were limitations to this study. Because it was a cadaver model the effect of healing could not be studied. The results suggest that proper healing is critical in restoring the ankle to its normal stability because stability was significantly decreased by cutting the ATFL and CFL but was not fully restored immediately after repairing both the ATFL and CFL. In addition, 18 or 20 specimens failed at the suture/soft tissue interface. Allowing time for the tendon to heal to the bone would protect the repair from this mode of failure. Another weakness of the study was the limitation of the mechanical testing. We were only able to test in one configuration and not simulate the dynamic muscle and ground reaction forces the foot and ankle encounter in vivo.

However, the most common mode of injury is inversion, plantar flexion, and internal rotation .[10] That is the orientation used to test the ankles in the current study.

CONCLUSION

This study found a greater increase in stiffness following combined ATFL/CFL repair compared to ATFL-only repair, although this did not reach statistical significance. The CFL fails before the ATFL, potentially indicating its vulnerability immediately following repair.
Restoring the CFL likely plays a relevant role in lateral ligament repair, however sufficient time for ligament healing should be allowed before rehabilitation stresses are applied. In addition, the repairs predominantly failed at the suture/soft tissue interface which indicates a potential need to develop a better interface.

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