Ultrasound-Guided Anterior Talofibular Ligament Repair With Augmentation Can Restore Ankle Kinematics

A Cadaveric Biomechanical Study

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Background: Anterior talofibular ligament (ATFL) repair of the ankle is a common surgical procedure. Ultrasound (US)-guided anchor placement for ATFL repair can be performed anatomically and accurately. However, to our knowledge, no study has investigated ankle kinematics after US-guided ATFL repair.

Hypothesis: US-guided ATFL repair with and without inferior extensor retinaculum (IER) augmentation will restore ankle kinematics.

Study Design: Controlled laboratory study; Level of evidence, 4.

Methods: A 6 degrees of freedom robotic testing system was used to apply multidirectional loads to fresh-frozen cadaveric ankles (N = 9). The following ankle states were evaluated: ATFL intact, ATFL deficient, combined ATFL repair and IER augmentation, and isolated US-guided ATFL repair. Three loading conditions (internal-external rotation torque, anterior-posterior load, and inversion-eversion torque) were applied at 4 ankle positions: 30° of plantarflexion, 15° of plantarflexion, 0° of plantarflexion, and 15° of dorsiflexion. The resulting kinematics were recorded and compared using a 1-way repeated-measures analysis of variance with the Benjamini-Hochberg test.

Results: Anterior translation in response to an internal rotation torque significantly increased in the ATFL-deficient state compared with the ATFL-intact state at 30° and 15° of plantarflexion ($P = .022$ and .03, respectively). After the combined US-guided ATFL repair and augmentation, anterior translation was reduced significantly compared with the ATFL-deficient state at 30° and 15° of plantarflexion ($P = .0012$ and .005, respectively). Anterior translation was not significantly different for the isolated ATFL-repair state compared with the ATFL-deficient or ATFL-intact states at 30° and 15° of plantarflexion.

Conclusion: Combined US-guided ATFL repair with augmentation of the IER reduced lateral ankle laxity due to ATFL deficiency. Isolated US-guided ATFL repair did not reduce laxity due to ATFL deficiency, nor did it increase instability compared with the intact ankle.

Clinical Relevance: US-guided ATFL repair with IER augmentation is a minimally-invasive technique to reduce lateral ankle laxity due to ATFL deficiency. Isolated US-guided ATFL repair may be a viable option if accompanied by a period of immobilization.

Keywords: ultrasound-guided surgery; chronic ankle instability; anterior talofibular ligament repair; augmentation; kinematics

Ankle sprains are the most frequent sports injuries, accounting for up to 30% of all sports injuries.[8,14] Of all ankle sprains, 85% involve the lateral ligament complex: in particular, the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL).[17] Despite nonoperative treatment after an ankle sprain, 20% of patients go on to develop chronic ankle instability[6] in which they experience some form of residual symptoms, such as recurrent ankle sprains, perceived instability, and continued pain.[11] When patients with chronic ankle instability do not recover with nonoperative treatment, surgical treatment is considered. Ligament repair of a torn ATFL or repair of the ATFL and

The Orthopaedic Journal of Sports Medicine, 10(8), 23259671221111397
DOI: 10.1177/23259671221111397

CFL has been performed instead of reconstruction with a graft when the quality of the remnants is viable.[28]

The open modified Broström-Gould technique is currently the most used surgical method for ATFL repair.[27,28] This technique allows for anatomic repair compared with older nonanatomic reconstruction techniques using the peroneal brevis tendon, but it is not without shortcomings. Nerve-related and wound complications after the open modified Broström-Gould procedure occur at a rate of 4.5% and 3.6%, respectively.[4] Arthroscopic repair has emerged as a less invasive treatment option over the past decade, resulting in comparable functional outcomes and patient satisfaction compared with open repair.[21] However, the nerve-related complication rate (5.2%) is as high as that for open surgery,[4] and the arthroscopic repair is challenging because the fibular attachment of the ATFL is not fully visible with arthroscopy.[25]

Musculoskeletal ultrasound (US) has gained popularity in sports medicine as both a diagnostic tool and an interventional tool. US-guided interventions are known to be more accurate than unguided interventions.[7] Our recent investigation demonstrated that US-guided anchor placement for ATFL repair was as anatomic as open ATFL repair.[13] However, to our knowledge, the kinematic response of the ankle to external loads after US-guided ATFL repair has not been assessed.

The aim of the current study was to evaluate the kinematics of the ankle joint after US-guided ATFL repair, with and without inferior extensor retinaculum (IER) augmentation, compared with the kinematics of intact and ATFL-deficient ankles. Our hypothesis was that US-guided ATFL repair with and without augmentation would restore the kinematics of the intact ankle.

METHODS

We used 14 fresh-frozen cadaveric ankles in this study, which received ethics committee approval. The specimens were stored at –20°C and thawed overnight at room temperature before testing. Ultrasonography was used to evaluate specimens for any change in ATFL, and radiography was used to assess bony morphology such as trauma, surgery, deformity, and arthritic change. Specimens with a thin ATFL (<1 mm as shown on US), skin pathology, or bony deformities were excluded from this study. Overall, 5 specimens were excluded as a result of the radiographic and US screening.

Included were 9 ankles with intact ATFL (100% male; mean age, 56 ± 17 years). A power analysis based on a previously published studies[15,24] specified that a minimum of 9 specimens were required for the detection of a 3° change in the inversion angle with an SD of 2.2°. This sample size calculation was performed using statistical software (EZR Version 2.13.0; Saitama Medical Center, Jichi Medical University) is based on R (http://www.r-project.org) and R Commander (http://socialsciences.mcmaster.ca/jfox/Misc/Rcmdr).

The lower leg was cut to 25 cm in length from the distal end of the medial malleolus, and the soft tissues, including skin, subcutaneous tissues, and muscles up to 5 cm superior to the medial malleolus, were carefully removed to expose the tibia and fibula. The soft tissues 4 cm from the tip of the calcaneus were removed, and three 4.5 mm–diameter wood screws were inserted into the calcaneus. The soft tissues 3 cm from the end of the fibula were left intact to preserve the ATFL and CFL. The exposed proximal tibiofibular syndesmosis was fixed with 2 screws of 4.5-mm diameter. After the tibial side was potted in an epoxy compound (Bondo; 3M), the calcaneus and attached screws were potted in a cylindrical mold (Figure 1).[19]

The ankle was mounted onto a 6 degrees of freedom (6DOF) robotic testing system (model FRS2010; MJT). The position and orientation repeatability of the robotic testing system were less than ±0.015 mm and ±0.01°, respectively. The sole of the foot faced upward. The tibial and calcaneal cylinders were secured to the clamps of the 6DOF robotic system. The tibial clamp was rigidly fixed to the lower plate of the robotic testing system, and the calcaneal clamp was attached to the upper end plate of the robotic manipulator through a universal force/moment sensor (model SI-660-60; ATI Delta IP60), which was used to provide feedback to the controller (Figure 2).[19]

The measurement uncertainty of the universal force/moment sensor was approximately 1% of full scale. The system was controlled by a LabVIEW program (Technology Services Inc) designed for ankle biomechanical testing and was operated in hybrid velocity-impedance control.

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Final revision submitted April 1, 2022; accepted May 12, 2022.

One or more of the authors has declared the following potential conflict of interest or source of funding: S.H. received a research grant from the Japanese Society of Orthopedic Ultrasonics for completion of this study. K.O. has received grant support from DJO and education payments from Arthrex. M.V.H. has received consulting fees from Zimmer Biomet and honoraria from In2Bones. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.

Ethical approval for this study was obtained from the University of Pittsburgh.

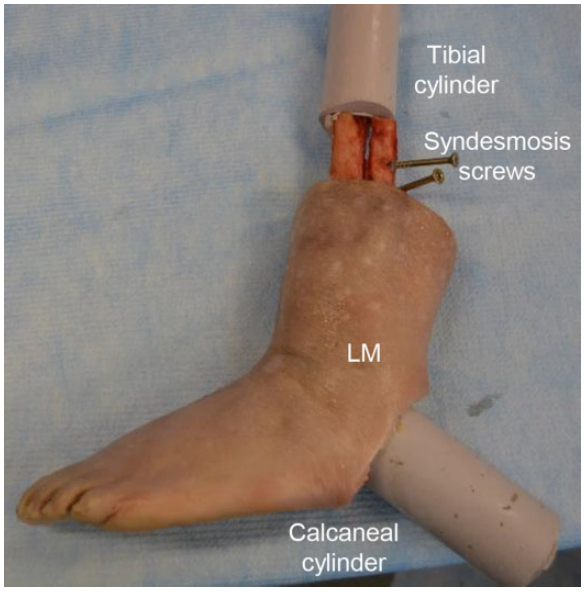

Figure 1. Left ankle specimen. After the skin and soft tissues 3 cm from the lateral malleoli (LM) were removed, the tibial and calcaneal screws were potted in a cylindrical mold. The tibiofibular syndesmosis was fixed with 2 screws.

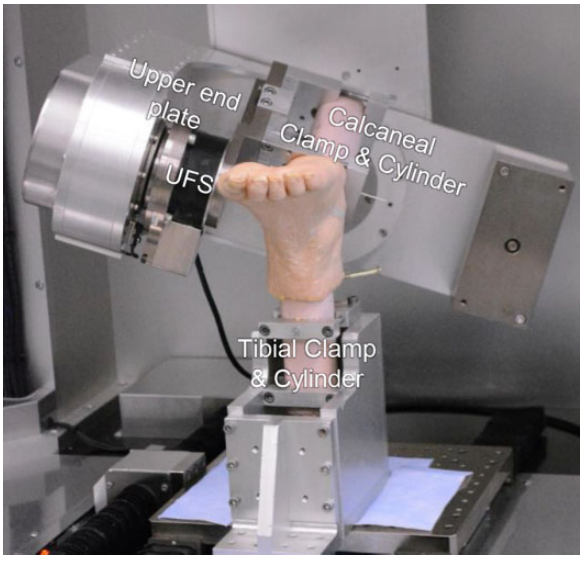

Figure 2. The 6 degrees of freedom robotic testing system. The tibial clamp and cylinder were fixed to the lower plate. The calcaneal clamp and cylinder were secured to the upper end plate with a universal force/moment sensor (UFS).

The tips of the lateral and medial malleoli were marked to define the ankle joint coordinate system in accordance with International Society of Biomechanics (ISB) recommendations.[26] The neutral configuration of the ankle joint complex defined by the ISB coordinate system used 3 planes: neutral dorsiflexion-plantarflexion, neutral inversion-eversion, and neutral internal rotation–external rotation. Neutral dorsiflexion-plantarflexion was defined as 0° between the plantar aspect of the foot and the line perpendicular to the long axis of the tibia projected on the sagittal plane of the ankle. Neutral inversion-eversion was defined as 0° between the plantar aspect of the foot and the line perpendicular to the long axis of the tibia projected on the frontal plane of the ankle. Neutral internal rotation–external rotation was defined as 0° between the frontal plane of the tibia and the line perpendicular to the long axis of the second metatarsal.[26]

The following 4 ankle states were evaluated: ATFL intact, ATFL deficient, ATFL repair combined with IER augmentation, and isolated ATFL repair. The 6DOF robotic testing system was used to apply 3 loading conditions to the ankle in each state: (1) 40-N anterior-posterior load, (2) 1.7-N·m internal-external rotation torque, and (3) 1.7-N·m inversion-eversion torque. A 5-N axial load was applied to maintain contact with the talocrural and subtalar joints during the application of the loads. During the anterior load, motion along the anterior-posterior axis was under displacement control. The medial-lateral, internal rotation–external rotation, and inversion-eversion axes were set under force control and allowed unconstrained motion to minimize forces and moments along each respective axis. The plantarflexion-dorsiflexion axis was constrained under position control. During the internal-external rotation and inversion-eversion torque, the internal-external rotation and inversion-eversion axes were under displacement control. The remaining axes were under force control except for the plantarflexion-dorsiflexion axis, which was constrained under position control.

The resultant kinematic parameters were recorded at 4 ankle positions: 30° of plantarflexion, 15° of plantarflexion, 0° of plantarflexion, and 15° of dorsiflexion.[22,24] We measured the maximum anterior-posterior translation in response to an anterior-posterior load, internal-external rotation in response to an internal-external rotation torque, inversion-eversion in response to an inversion-eversion torque, and anterior-posterior translation in response to internal-external rotation torque from the passive path. The passive path positions were defined as the positions of the ankle joint from 30° of plantarflexion to 15° of dorsiflexion when the forces and moments were minimized. We applied 20 N of anterior load and 0.85 N·m of internal-external rotation torque 5 times as preconditioning to avoid creep effects before each recording.

After data on the ATFL-intact state were acquired, the ATFL was sharply transected at the fibular attachment under direct arthroscopic visualization to create an ATFL-deficient state using standard anteromedial and anterolateral portals.[9] The complete transection of the ATFL was confirmed under arthroscopy as well as US. The loading conditions were applied again, and the resultant kinematic values were recorded for the ATFL-deficient ankle.

US-Guided ATFL Repair With and Without Augmentation

US-guided ATFL repair was performed using a high-frequency linear transducer of 18 MHz (HS1 Konica-

Minolta). For suture passage, a large spinal needle with a curved tip (MicroSutureLasso Minor Bend; Arthrex) was used (Figure 3).

The safe zone was delineated at 30° of plantarflexion with a marking pen between the dorsal cutaneous nerve of the superficial peroneal nerve and peroneal brevis to avoid iatrogenic superficial peroneal nerve and sural nerve injuries.[1] The talar stump of the cut ATFL was visualized in the short-axis view of the ATFL over the talar cartilage, just proximal to the talar attachment of the ATFL. Under US guidance, the curved tip of the MicroSutureLasso Minor Bend was introduced into the ATFL stump.[12] After the ATFL, subcutaneous tissue, and skin were penetrated, a nitinol loop wire was deployed. Next, upon visualization of the long axis of the ATFL, the MicroSutureLasso Minor Bend was inserted into the talar stump of the ATFL. The second needle was placed perpendicular to and below the first nitinol wire so that the 2 wires crossed (Figure 4). The suture configuration was similar to the modified Mason-Allen stitch.[23]

Figure 3. MicroSutureLasso Minor Bend (Arthrex) with a 30° curved tip. The lengths of the tip and shaft were 24 and 153 mm, respectively.

A bony peak of the fibular footprint of the ATFL was observed upon US, and an 18-gauge spinal needle was inserted at the footprint for marking. A 5-mm skin incision was made 10 mm distal-medial to the footprint, and the subcutaneous tissue was dilated. A suture anchor (DEX Fibertak; Arthrex) was placed at the anatomic fibular footprint after insertion of the drill guide followed by drilling under US guidance.[12] The 4 limbs of the nitinol wires were retrieved subcutaneously through the anchor incision below the superior peroneal nerve and above the ATFL. Each nitinol loop was relayed to each limb of the suture anchor, and 2 limbs of the suture anchor were clamped.

The long axis of the IER was visualized at its calcaneal attachment proximal to the peroneal tendons. The MicroSutureLasso Minor Bend was introduced into the IER and advanced within the safe zone to achieve augmentation.[10] After the skin was penetrated, a nitinol loop wire was deployed (Figure 5, A and B). A second suture anchor was placed 1 cm proximal to the first anchor in the same fashion under US guidance. After retrieval of the nitinol wire through the anchor incision, one limb of the suture anchor were relayed by the nitinol loop and clamped. Pretensioning was applied manually to both suture anchors for ATFL repair and those for augmentation at 0° of plantarflexion,

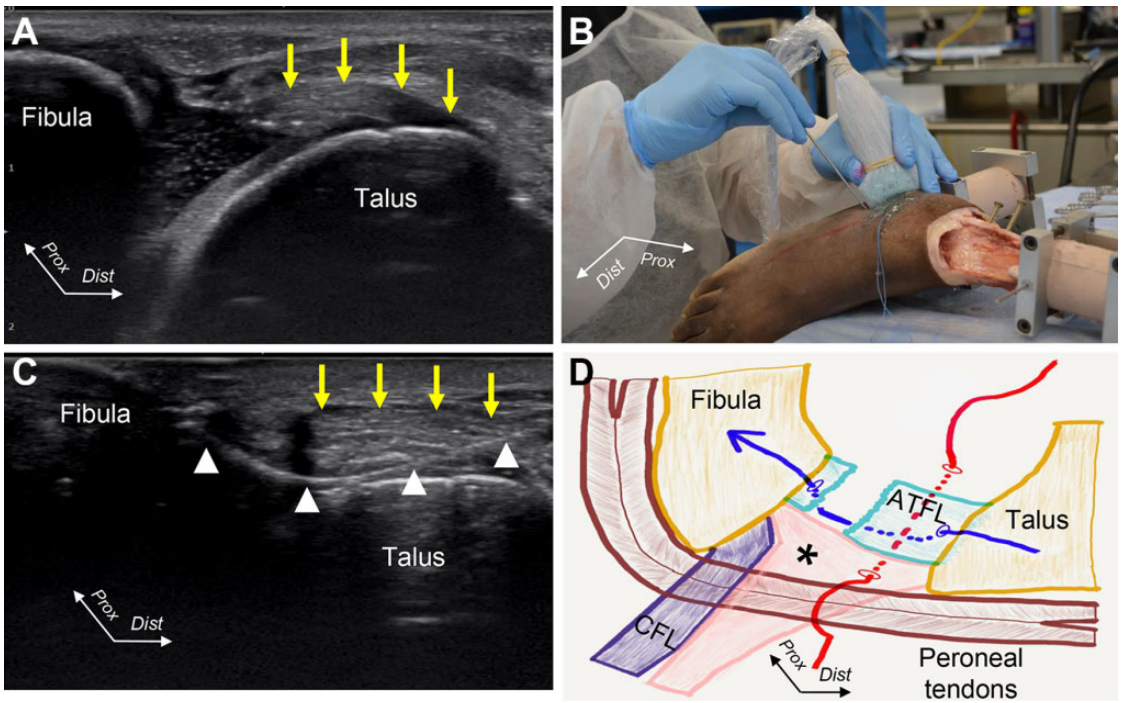

Figure 4. Ultrasound (US)-guided repair of the anterior talofibular ligament (ATFL). (A) The talar stump of the ATFL was visualized (yellow arrows) in the long axis view. (B and C) A MicroSutureLasso Minor Bend (white triangles) was introduced into the talar stump of the ATFL (yellow arrows) under US guidance. (D) The first suture (red line) penetrated the ATFL and the connecting fiber (black asterisk) between the ATFL and the calcaneofibular ligament (CFL). The second suture (blue line) passed through the long axis of the ATFL below the first suture. The 2 sutures were crossed perpendicularly. Dist, distal; Prox, proximal.

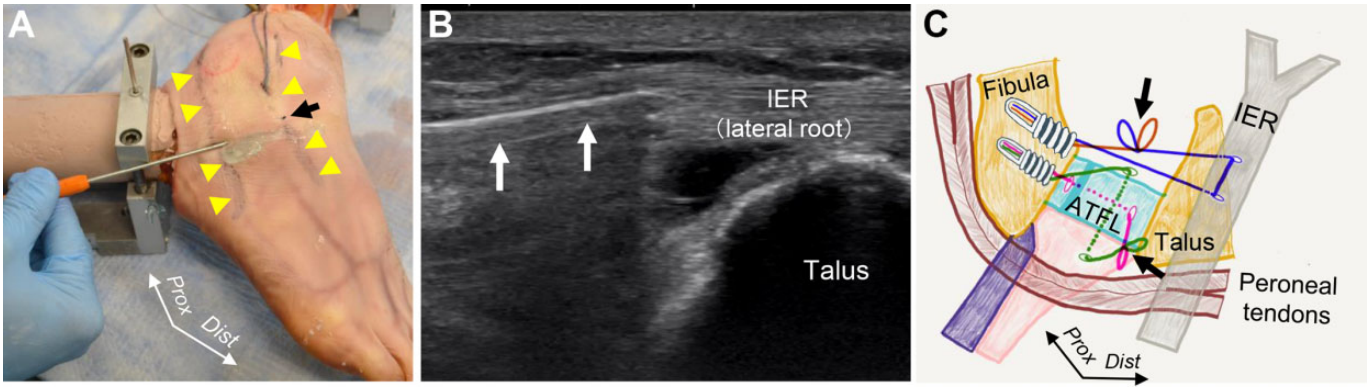

Figure 5. Combined ultrasound (US)-guided anterior talofibular ligament (ATFL) repair and augmentation with inferior extensor retinaculum (IER). (A and B) A MicroSutureLasso Minor Bend (white arrows) was introduced into the IER lateral root under US guidance. The tip of the needle (black arrow) penetrated the skin within the safe zone (yellow arrowheads). (C) Schematic diagram of combined US-guided ATFL repair and augmentation with IER. Surgical clamps were applied to each pair of suture anchors at the knot area (black arrows). Dist, distal; Prox, proximal.

and surgical clamps were put on each pair of suture anchors (Figure 5C).

The loading conditions were then applied to the combined ATFL repair and augmentation, and the resultant kinematic values were recorded. The clamp of the augmentation repair was released to test the isolated ATFL-repair state. The loading conditions were then applied to the isolated ATFL repair, and the resultant kinematic values were recorded. Dissection after the testing protocol confirmed anatomic anchor placement and suture passage in all specimens.

Statistical Analysis

A 1-way repeated-measures analysis of variance was used to compare the kinematics among each group using SAS Version 9.4. Post hoc comparisons of the differences were performed between the (1) ATFL-deficient and ATFL-intact states, (2) ATFL-deficient and combined ATFL-repair and augmentation states, (3) ATFL-deficient and isolated ATFL-repair states, and (4) ATFL-intact and isolated repair states. The Benjamini-Hochberg procedure was used for the post hoc analysis, and statistical significance was set at $P < .05$.

RESULTS

Internal Rotation Torque

Anterior translation in response to internal rotation torque at 30° of plantarflexion and 15° of plantarflexion increased by 85% (from 3.3 to 6.1 mm; $P = .022$) and 80% (from 3.0 to 5.4 mm; $P = .03$), respectively, between the intact and ATFL-deficient states (Figure 6A). Internal rotation in response to internal rotation torque at 30° and 15° of plantarflexion increased by 41% (from 13.7° to 19.3°) and 34% (from 11.8° to 15.8°), respectively, after ATFL injury, although this was not significant ($P = .059$ and .18, respectively) (Figure 6B). No statistically significant differences between the intact and ATFL-deficient states were observed in anterior translation ($P = .35$) or internal rotation ($P = .56$) at 0° of plantarflexion.

Because internal rotation load at 30° of plantarflexion and 15° of plantarflexion were the most optimal positions to detect lateral ankle laxity, the kinematic values at this loading condition and ankle positions were the ones mainly examined in the following analyses. Other statistical results are summarized in Table 1.

After the US-guided ATFL repair with IER augmentation, anterior translation at 30° and 15° of plantarflexion decreased from the ATFL-deficient state by 74% (from 6.1 to 1.6 mm; $P = .0012$) and 63% (from 5.4 to 2.0 mm; $P = .005$), respectively. Internal rotation at 30° and 15° of plantarflexion decreased by 46% (from 19.3° to 10.4°; $P = .0012$) and 25% (from 15.8° to 11.8°; $P = .057$), respectively. After the augmentation clamp was released, no statistically significant differences between the isolated ATFL-repair state and ATFL-deficient state were observed in anterior translation at 30° and 15° of plantarflexion ($P = .16$ and .34, respectively) or internal rotation at 30° and 15° of plantarflexion ($P = .30$ and .18, respectively).

No statistically significant differences between the intact and isolated ATFL-repair states were seen in anterior translation at 30° and 15° of plantarflexion (both $P = .35$) or in internal rotation at 30° and 15° of plantarflexion ($P = .5$ and .97, respectively).

Anterior Load

The anterior load resulted in no statistically significant change in anterior translation between the intact and ATFL-deficient states at any ankle positions, although anterior translations decreased by 73% at 30° of plantarflexion ($P = .016$) and by 63% at 15° of plantarflexion ($P = .016$) after the US-guided ATFL repair with IER augmentation (Figure 7).

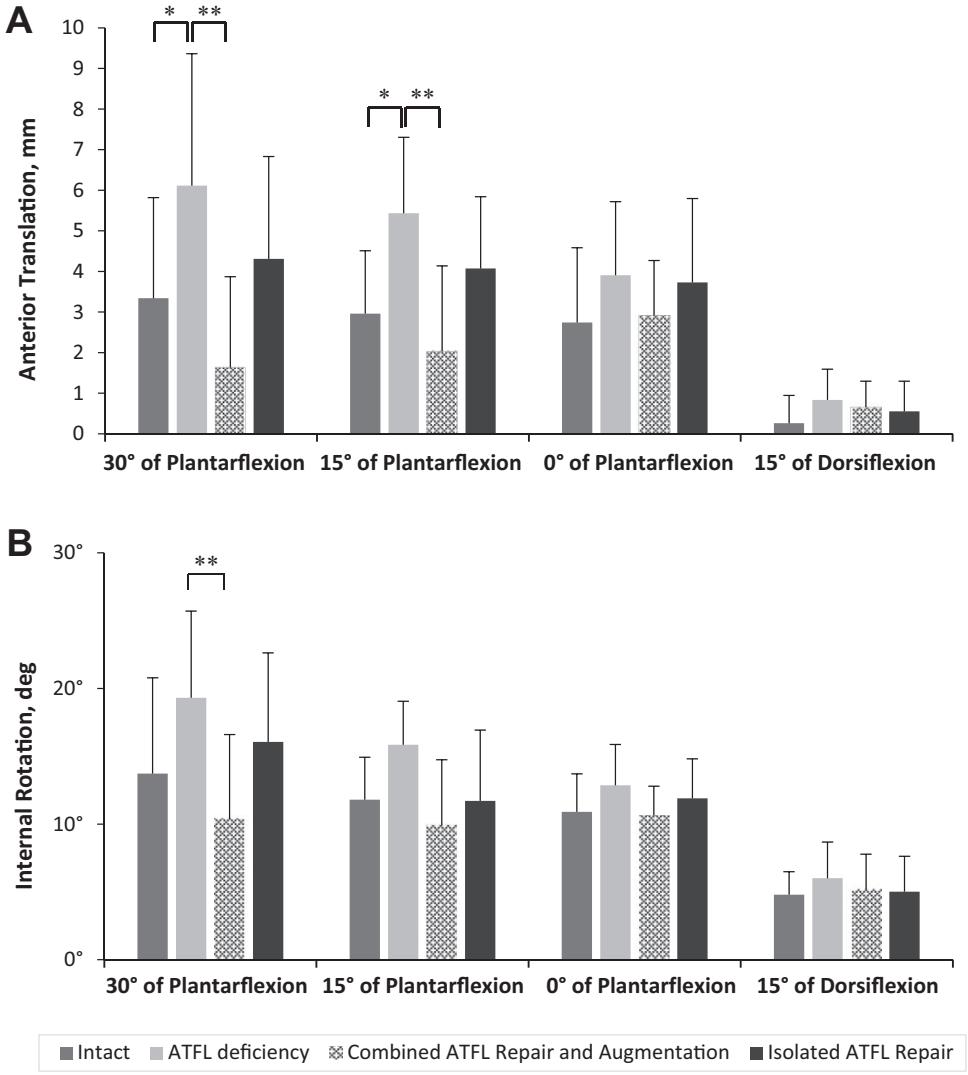

Figure 6. Mean (A) anterior translation and (B) internal rotation in response to 1.7-N·m internal rotation torque. Error bars represent SDs. Significant difference between *intact versus anterior talofibular ligament (ATFL) deficiency ($P < .05$) and **combined ATFL repair with augmentation versus ATFL deficiency ($P < .05$).

No statistically significant differences between the ATFL-deficient and isolated ATFL-repair states were observed in anterior translation at 30° and 15° of plantarflexion ($P = .39$ and .39, respectively). As well, no statistically significant differences between the isolated ATFL-repair and intact states were found in anterior translation at 30° and 15° of plantarflexion ($P = .55$ and .98, respectively).

Inversion Torque

The inversion torque resulted in no statistically significant change in inversion displacement between the intact and ATFL-deficient states at any ankle positions, although a significant reduction of displacement was observed at 30° of plantarflexion ($P = .041$) after the US-guided ATFL repair with IER augmentation. Other results are summarized in Table 1.

DISCUSSION

The most important findings in the current study were that (1) US-guided ATFL repair with IER augmentation resulted in a significant reduction in lateral ankle laxity in response to internal rotation torque due to ATFL deficiency and (2) the isolated ATFL-repair state in response to internal rotation torque showed no significant difference from either the ATFL-deficient state or the intact state.

The most optimal loading conditions and ankle positions to detect lateral ankle instability due to isolated ATFL deficiency in this study were internal rotation torque at 15° and 30° of plantarflexion, where a statistically significant

difference between the intact state and ATFL-deficient state was observed in anterior translation. An inversion torque of 5.7 N·m and an anterior load of 80 N were previously applied to an isolated ATFL-deficient state, and an isolated ATFL injury caused only a small magnitude of change in laxity (<4° and 2 mm, respectively).[2] Similarly, the current study identified only a small amount of displacement after an anterior load was applied to the ATFL-deficient state; meanwhile, a significantly larger displacement was recorded when internal rotation torque at 15° and 30° of plantarflexion was applied. This result agreed with another biomechanical study that compared nonanatomic and anatomic ATFL repair and found a statistically significant difference between the 2 conditions only when internal rotation torque was applied at 15° and 30° of plantarflexion.[24] In the current study, anterior translation in response to an internal rotation torque showed a statistically significant difference between the ATFL-deficient and intact ankle. According to a biomechanical study, the anterolateral drawer test, which allowed unconstrained internal rotation of the ankle, provoked almost twice the talus displacement as the simple anterior drawer test, in which the ankle was allowed to move only anteriorly in response to anterior load.[18] In the current study, unconstrained anterior translation during internal rotation torque led to more anterior translation than anterior load and therefore resulted in a higher anterior translation at 15° and 30° of plantarflexion.

In the current study, US-guided ATFL repair with IER augmentation significantly reduced anterior translation at 15° and 30° of plantarflexion in response to an internal rotation torque. Behrens et al[3] compared open Broström-Gould repair (namely, ATFL and CFL repair with IER augmentation) with ATFL- and CFL-deficient states using cadavers and a video motion analysis system and concluded that open Broström-Gould repair reduced the laxity due to lateral ligament deficiency. Although all procedures in the current study were performed under a minimally-invasive technique using US, a similar reduction of lateral ankle laxity was

TABLE 1
P Values After Post Hoc Comparisons Using the Benjamini-Hochberg Procedure[a]

		P Values		
Load	Displacement	30° of PF	15° of PF	0° of PF
ATFL Deficient vs Intact				
IR	Anterior	.022[b]	.03[b]	.31
IR	IR	.059	.18	.52
Anterior	Anterior	.17	.39	.52
Inversion	Inversion	.65	.65	.78
ATFL Deficient vs Augmented ATFL Repair				
IR	Anterior	.001[b]	.005[b]	.35
IR	IR	.001[b]	.057	.46
Anterior	Anterior	.016[b]	.016[b]	.52
Inversion	Inversion	.041[b]	.61	.8
ATFL Deficient vs Isolated ATFL Repair				
IR	Anterior	.16	.34	.35
IR	IR	.3	.18	.73
Anterior	Anterior	.39	.39	.52
Inversion	Inversion	.65	.86	.78
Isolated ATFL Repair vs Intact				
IR	Anterior	.35	.35	.35
IR	IR	.5	.97	.73
Anterior	Anterior	.55	.98	.98
Inversion	Inversion	.96	.8	.85

[a]ATFL, anterior talofibular ligament; IR, internal rotation; PF, plantarflexion.
[b]Statistically significant difference between comparison groups ($P < .05$).

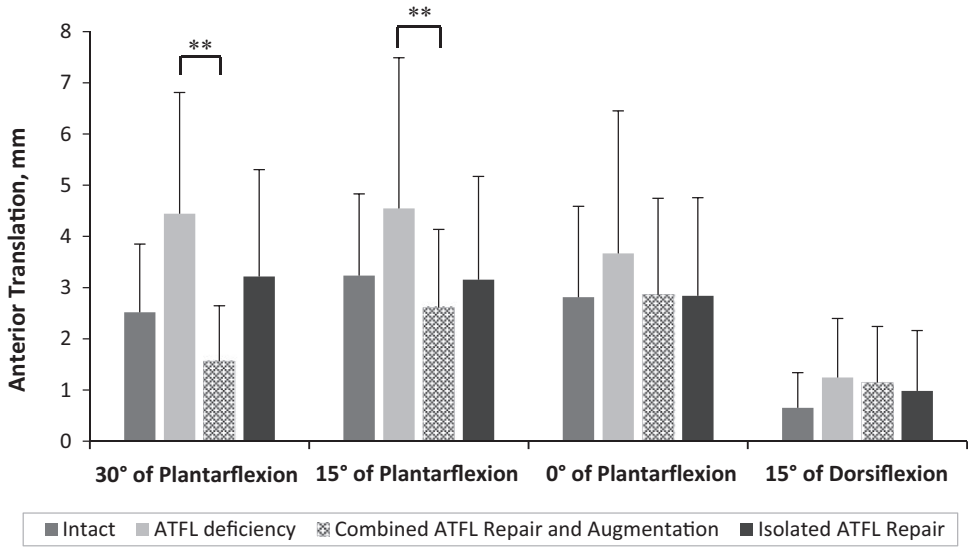

Figure 7. Mean anterior translation in response to 40-N anterior load. Error bars represent SDs. **Significant difference between combined anterior talofibular ligament (ATFL) repair with augmentation versus ATFL deficiency ($P < .05$).

observed after US-guided ATFL repair with IER augmentation.

In the current study, isolated ATFL repair was not significantly different compared with the ATFL-deficient state or the intact state. A previous biomechanical study using a 6DOF robotic testing system compared ATFL- and CFL-deficient states versus Broström repair (ie, ATFL and CFL repair) states.[20] The investigators found a significant difference between the lateral ligament-deficient and repaired states, as opposed to our result. In their testing protocol, the Broström repair state was tested before the augmented Broström repair state, and the investigators did not perform any preconditioning to prevent sutures from cutting through the repaired ligament. In contrast, we tested the isolated ATFL repair after the augmented ATFL repair and performed preconditioning before each test to minimize creep effects. The tissue fatigue of the isolated ATFL repair due to sequential testing together with the preconditioning could elucidate why our study did not show a difference between the ATFL-deficient and isolated ATFL-repair states.

Other previous studies using a 6DOF robot compared "intact" versus repaired states rather than "deficient" versus repaired states. Larkins et al[16] compared intact states with ATFL- and CFL-repair states and found that ATFL- and CFL-repair states had increased laxity compared with the intact state. Those investigators concluded none of the repair methods at time zero restored kinematics, and they suggested a period of immobilization for biological healing. In contrast, Shoji et al[24] compared anatomic and nonanatomic ATFL-repair states with an intact state and found no significant difference between intact and anatomic ATFL repair. Similar to that study, our study demonstrated no significant difference between intact and isolated ATFL-repair states. When the intact state was compared with the repaired states, Larkins et al[16] showed a significant difference, whereas our study did not. This conflicting result may derive from the difference in loading conditions. Whereas Larkins et al[16] applied anterior force of 88 N and inversion torque of 5 N·m, we applied 40-N anterior force, 1.7-N·m rotational torque, and 1.7-N·m inversion torque. A high load can make sutures cut through the repaired ligament in cadavers. Therefore, in the clinical setting, loading more than 40-N anterior force, 1.7-N·m rotational torque, and 1.7-N·m inversion torque on the US-guided ATFL repair should be avoided at time zero before any biological healing occurs. The isolated US-guided ATFL repair may still be a viable option if accompanied by a period of immobilization to protect it from high loading and promote biological healing.

Limitations

There were limitations in the current study. The sample size of this study was small, and the study may have been underpowered for some comparisons. This was a biomechanical study that did not fully replicate the clinical setting. The ATFL was sectioned on the fibular side of the ligament to create the ATFL-deficient state. Neither talar-side ATFL tears nor chronic ATFL tears in which the ligament is elongated were included in the current study; however, most ATFL tears are located on the fibular side.[5] The specimens with a thin ATFL (<1 mm based on ultrasonographic measurement) were excluded, and patients with a severely attenuated ATFL are not good candidates for this procedure. We could not use differential variable reluctance transducers placed over the ATFL to test the displacement directly, because we preserved the skin and subcutaneous tissues when we performed and tested the US-guided ATFL repair. There could have been motion of the subtalar joint that could not be detected in this experiment. The suture anchors were not tied by knots but were clamped for repetitive use of anchors. The fatigue of clamps and anchors could increase the laxity of the repaired ankles. Finally, US-guided ATFL repair with augmentation requires specific skills pertaining to both ultrasonography and ATFL repair, and therefore the results may vary depending on the skills of the operator.

CONCLUSION

The current biomechanical study demonstrated that combined US-guided ATFL repair and augmentation could reduce lateral ankle laxity due to ATFL deficiency. The isolated US-guided ATFL repair did not decrease the laxity due to ATFL deficiency or lead to instability compared with the intact ankle. Future research will need to assess the feasibility of US-guided ATFL repair and augmentation and the clinical outcomes after this procedure.

ACKNOWLEDGMENT

The authors thank Clair N. Smith and Hiroki Matsui for helping with the biostatistical analysis of the manuscript.

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