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In vivo cartilage contact strains in patients with lateral ankle instability

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Abstract

Damage to the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) during ankle sprain may be linked to the development of osteoarthritis. Altered tibiotalar kinematics have been demonstrated in these patients, but the effects of lateral ankle instability (LAI) on in vivo cartilage strains have not been described. We hypothesized that peak cartilage strains increase, and the location is shifted in patients with ATFL injuries. We used 3-D MRI models and biplanar fluoroscopy to evaluate in vivo cartilage contact strains in seven patients with unilateral LAI. Subjects had chronic unilateral ATFL injury or combined ATFL and CFL injury, and were evaluated with increasing load while stepping onto a force plate. Peak cartilage strain and the location of the peak strain were measured using the contralateral normal ankle as a control. Ankles with LAI demonstrated significantly increased peak strain when compared with ATFL-intact controls. For example, at 100% body weight, peak strain was $29 \pm 8\%$ on the injured side compared to $21 \pm 5\%$ on the intact side. The position of peak strain on the injured ankle also showed significant anterior translation and medial translation. At 100% body weight, the location of peak strain in the injured ankle translated anteriorly by $15.5 \pm 7.1\text{mm}$ and medially by $12.9 \pm 4.3\text{mm}$ relative to the intact ankle. These changes correspond to the region of clinically-observed osteoarthritis. Chronic LAI, therefore, may contribute to the development of tibiotalar cartilage degeneration due to altered cartilage strains.

Introduction

Ankle sprains are a common problem, resulting in 75% of all ankle injuries and 25% of injuries in running and jumping sports (DiGiovanni and Brodsky 2006, Mack 1982). Although ankle sprains heal with conservative treatment 60-80% of the time (Hintermann et al. 2002), lateral ankle instability (LAI) can result when these injuries fail to heal. Morbidity affecting work and athletic performance may result from LAI, and progression to osteoarthritis (OA) can be a long term concern (Brostrom and Sundelin 1966, Harrington 1979, Komenda and Ferkel 1999). Lateral ligament damage, which most commonly includes injury to the anterotalofibular ligament (ATFL) and often to the calcaneofibular ligament (CFL), has been shown to be the main instigator in development of ligamentous

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posttraumatic arthritis (Valderrabano et al. 2006). The development of osteoarthritis in patients with longstanding LAI is thought to be as high as 78% beyond 10 years (Harrington 1979, Lofvenberg et al. 1994, Povacz et al. 1998).

Altered tibiotalar kinematics resulting from lateral ligamentous laxity or rupture has been hypothesized to contribute to the development of chondral lesions and arthritis (Hirose et al. 2004, Omori et al. 2004, Valderrabano et al. 2006). Disruption of the ATFL increases the anterior translation (Caputo et al. 2009, Hollis et al. 1995, Hollis et al. 1995, Ringleb et al. 2005) and internal rotation (Caputo et al. 2009, Stormont et al. 1985) of the talus relative to the tibia. Although a causal link between LAI and osteoarthritis has yet to be established, altered stress distributions have been theorized to contribute to cartilage damage (Hirose et al. 2004, Omori et al. 2004, Valderrabano et al. 2006, Van Dijk CN 1996). Clinical observations of degenerative changes in the medial side of the joint are consistent with altered kinematics, because internal rotation and anterior translation might cause medial abutment and increased cartilage strain between the talus and medial malleolus (Caputo et al. 2009, Harrington 1979, Krips et al. 2001, Van Dijk CN 1996).

Despite these findings, there are limited data on the in vivo cartilage contact strain patterns in patients with chronic LAI. In this study, we used 3-D MRI modeling combined with biplanar fluoroscopy (Caputo et al. 2009, DeFrate et al. 2004) to evaluate the cartilage contact strain distribution of the talocrural joint in patients with LAI refractory to conservative management. We hypothesized that peak cartilage contact strains would increase and the location of peak strain would demonstrate an anteromedial shift in the injured ankles of patients with LAI relative to their normal ankle.

Materials and Methods

Patient Recruitment

Seven patients (4 female, 3 male, mean age 45 years, range 33 to 57 years) with unilateral chronic LAI and a healthy contralateral ankle were recruited consecutively from clinic after obtaining informed consent for this IRB approved protocol. All subjects had failed conservative treatment for at least 6 months and had complaints of pain, instability, and decreased proprioception. Clinical examination was performed by faculty foot and ankle orthopedic surgeons to confirm the diagnosis of chronic LAI. Additionally, magnetic resonance images (MRI) were obtained to corroborate clinical findings of ATFL injury. Because subjects with isolated ATFL injury can be difficult to recruit, subjects with combined ATFL and CFL injuries were also included in the study (Caputo et al. 2009). Three subjects had combined ATFL and CFL injuries, while four had isolated ATFL injuries. Subjects were excluded if they had a history of previous foot and ankle surgery or had MRI or surgical findings of additional ankle pathology. Reasons for exclusion included peroneal tendinopathy, intra-articular ossicles, or evidence of cartilage degeneration.

Imaging

MR images were obtained using a 3.0T magnet (Trio Tim, Siemens, Germany) and an 8 channel receive-only foot and ankle coil (InVivo, Orlando, FL). Both ankles were imaged in the sagittal plane separately using a 3D double-echo steady state sequence (DESS, flip angle: 25°, TE: 6ms, TR: 17ms, field of view: 15×15 cm², matrix: 512×512 pixels², slice thickness: 0.7mm, resolution 0.29mm). Subjects were kept non-weightbearing for at least 30 minutes prior to the MRI scan. Each ankle was imaged twice, with and without water excitation, to allow for improved contrast between bone and soft tissue interfaces.

The images were then imported into solid modeling software (Rhino, McNeel and Associates, Seattle, WA) and the outlines of the tibia, talus, and cartilage surfaces were

traced in each MR slice (Figure 1). Mesh surfaces of the tibia, talus, and cartilage were created independently for the tibia and talus using solid modeling software (Geomagic Studio, Geomagic, Research Triangle Park, NC). While creating the 3-D models, the investigators were blinded to the results of the clinical MR and had no knowledge of which ankle was injured.

After acquiring the MR images, subjects were imaged while stepping onto a force plate using biplanar fluoroscopy (Pulsera, Philips, The Netherlands). The order in which the intact and injured ankles were tested was alternated between patients. The fluoroscopes recorded simultaneous images of the ankle at a resolution of 1024×1024 pixels² from the anteromedial and anterolateral directions. With appropriate lead protection, the subject progressively placed 25%, 50%, 75% and 100% body weight on the foot (Caputo et al. 2009), while keeping the shank perpendicular to the ground. Patients held each position for 3s as the image was recorded.

Strain calculation

Each pair of fluoroscopic images was imported into solid modeling software and positioned in a 3-D environment to reproduce the orientation of the fluoroscopes during testing. The bony outlines of the images were detected with a Canny filter using custom-written pixel density gradient matrix edge detection software (Mathematica 6.0, Wolfram Research Inc, Champaign, IL) (Defrate et al. 2006). The 3-D models of the tibia and talus were then imported into the software and individually manipulated in six degrees-of-freedom until the projections matched the bony outlines, reproducing the in vivo motion of the ankle joint (Caputo et al. 2009). Five matching trials were averaged to determine the relative motion of the tibia and talus for each pair of fluoroscopic images. A previous study from our laboratory found that this method is accurate to within 0.1mm and 0.3° of rotation (Caputo et al. 2009).

The cartilage surfaces were then imported separately and aligned with the tibial and talar models respectively. Since the surfaces were created in a non-weightbearing condition during MR scan, the registered tibia and talus cartilage mesh surfaces overlapped where these surfaces were in contact. Engineering strain across the talocrural joint was approximated by dividing the distance of overlap by the sum of the thicknesses of the undeformed talar and tibial cartilage layers (Cohen et al. 2003, Cohen et al. 2001, Wan et al. 2008) (Figure 1). The closest points between the two cartilage surfaces were used to determine the overlap distance. These calculations were performed using custom-written software (Mathematica 6.0) to create a cartilage strain map of the overlapping surface of the joint, including the location and magnitude of peak cartilage contact strain.

The location of the peak cartilage contact strain was described using a Cartesian coordinate system. The coordinate system was created on both ankles of each subject simultaneously using an iterative closest point technique (Caputo et al. 2009). The proximodistal axis was parallel to the long axis of a cylinder fit through the shaft of the intact tibia, the mediolateral axis was parallel to a line connecting the extremes of the tibia, and the anteroposterior axis was represented by a line orthogonal to both of these. The center of a circle fit to the curve of the talar dome defined the origin in the sagittal plane, and the geometric center of the talus defined the medial-lateral position. In this fashion, a coordinate system was established on the center of the talus to measure the position of the contact points in the mediolateral and anteroposterior directions (Figure 2). For statistical analysis, the Wilcoxon Ranked Sum Test was used to quantify differences in the magnitude and location of the peak contact strain between intact and ATFL deficient ankles. Differences were considered statistically significant where $p < 0.05$.

Validation of Contact Strain Measurements

In this study, strain was approximated by the ratio of the penetration distance to the cartilage thickness of the two cartilage layers (Cohen et al. 2003, Cohen et al. 2001, Wan et al. 2008). This results in an approximation of the average of the strain throughout the thickness of both cartilage layers. In order to determine how well this measure approximates the strain throughout the thickness of the talar cartilage layer, a validation study was performed.

First, we placed a previously-frozen cadaveric ankle into a custom-built non-metallic loading frame. Following the described MR protocol, we obtained images of the ankle in a relaxed position in the testing device with the cartilage minimally deformed. Three-dimensional models of the tibia, talus, and corresponding cartilage surfaces were created. We then applied an axial displacement of approximately 1mm to the tibiotalar joint using the loading frame. A second 3D model was made of these loaded ankle bones and cartilage surfaces.

Using these two models, we used an iterative closest point technique to align the bones of the unloaded cadaveric trial with the bones of the loaded trial. After registering the cartilage surfaces of the unloaded joint to the bones, the strain approximated from the overlap between the two cartilage layers was compared with that calculated from the direct measure of the talar cartilage thickness change before and after loading.

The strain approximated from the overlap of the two cartilage layers resulted in a similar pattern of contact strain as that measured in the talar cartilage directly from MRI (Figure 3). The peak strain predicted from the amount of overlap was 42%, compared to 48% for the strain measured directly from the loaded joint. The location of the peak strains were 0.9mm from each other. Although there were differences between these two strain measures, these results indicate that the average strain approximated from the overlap of the talar and tibial cartilage layers provides a useful approximation to the strain in the talus.

Repeatability of Peak Contact Strain Measurements

In order to evaluate the repeatability of our peak strain measurements, the healthy ankle of one of the subjects was tested on three separate days. For each trial, the subject was imaged using biplanar fluoroscopy while stepping onto the force plate, as described above. The fluoroscopic images were used to reproduce the motion of the ankle motion, and peak strain was calculated for each of the three trials. Peak cartilage contact strain at 100% body weight averaged $26 \pm 2\%$ (24, 26, and 27%) for the three trials. These results indicate that this methodology can reproducibly measure peak cartilage contact strains.

Results

Peak Cartilage Contact Strain

Lateral ankle instability altered the contact strain distributions compared to the contralateral intact ankle (Figure 4). The peak cartilage contact strain in both the deficient and intact ankles increased with increasing percentages of body weight (Figure 5). In the intact ankle, the strain increased from $16 \pm 4\%$ at 25% body weight to $21 \pm 4\%$ at 100% body weight. A similar trend of increasing peak contact strain with increasing body weight presented in the injured ankle. However, there was a statistically significant increase in contact strain in the injured ankle at 50% ($p = 0.04$), 75% ($p = 0.03$) and 100% ($p = 0.02$) body weight. In the injured ankle, peak strain ranged between $19 \pm 8\%$ (at 25% body weight) and $29 \pm 9\%$ (at 100% body weight).

Anteroposterior Translation of Peak Cartilage Strain

In the intact ankle, the anteroposterior position of peak cartilage contact strain remained between 8.3 ± 4.1 mm (at 100% body weight) and 5.3 ± 4.7 mm (at 50% body weight) posterior to the middle of the talar dome (Figure 6). However, in the injured ankle, increased anterior translation of the peak strain with increased body weight was observed. At 25% body weight, the peak strain was 3.6 ± 8.1 mm posterior to middle of the talar dome. At 100% body weight, the peak strain was 7.9 ± 2.7 mm anterior to middle of the talar dome. At 50%, 75%, and 100% body weight, this anterior translation demonstrated a statistically significant increase over the position of peak strain on the talus of the intact ankle ($p = 0.02$). At 100% body weight, the position of peak strain on the injured ankle was 15.5 ± 7.1 mm anterior to the position of peak strain on the intact talus.

Mediolateral Translation of Peak Cartilage Strain

In the intact ankle, increased loading caused a slight lateral translation of the peak strain (Figure 7). At 25% body weight, peak strain was located 2.0 ± 4.3 mm medial to the middle of the talus and translated to 2.3 mm (± 2.3 mm) lateral at 100% body weight. The medial position of the injured ankle ranged from 1.2 ± 8.8 mm at 25% body weight to 10.6 ± 4.3 mm at 100% body weight. At 100% body weight, there was a statistically significant increase in medial translation of the peak cartilage strain on the talus of the injured ankle when compared with the intact ankle ($p = 0.02$). The position of peak strain on the injured talus was located 12.9 ± 4.3 mm medial to the position of peak strain on the intact ankle.

Discussion

In this study, we used MR imaging and orthogonal fluoroscopy to determine the in vivo cartilage contact strains in patients with LAI. We found that LAI increased and shifted anteromedially the peak cartilage contact strain. The altered in vivo contact strains observed in this study might help to explain the degeneration observed on the anteromedial portion of the joint in patients with chronic LAI (Harrington 1979, Hintermann et al. 2002, Okuda et al. 2005, Valderrabano et al. 2006).

Our data can be explained by previous findings of increased anterior translation (Caputo et al. 2009, Hollis et al. 1995, Hollis et al. 1995, Ringleb et al. 2005) and internal rotation (Caputo et al. 2009, Stormont et al. 1985) of the talus with LAI. In the absence of the ATFL, the increased internal rotation and anterior translation likely shifts the contact anteromedially, due to the curved articular surface of the medial malleolus (Caputo et al. 2009). These findings are consistent with the data of the present study, as we observed an increase and anteromedial shift of the peak contact strain with LAI.

Previous cadaver studies have reported increased contact pressures on the medial side of the talus with sectioning of the ATFL (Omori et al. 2004, Rosenbaum et al. 1997). However, few previous studies have evaluated cartilage contact in the ankle joint under in vivo loading conditions. A recent study used MR and dual orthogonal fluoroscopic imaging to evaluate the real time in vivo contact deformation in normal ankle cartilage (Li et al. 2008). After 10s of loading, the peak cartilage strain in the talocrural joint was 26%. This value is comparable to our peak strain data of 21% in the intact ankle.

Altered loading after injury has been thought to be detrimental to normal cartilage health (Andriacchi et al. 2006, Elliott et al. 1999, Li et al. 2008). Our findings support previous literature linking LAI to the development of osteoarthritis, and may suggest that increased strain in the anteromedial region of the joint could lead to degenerative cartilage changes. One group studied 247 consecutive patients referred for ankle arthritis (Valderrabano et al. 2006). They determined that lateral ligament damage was the main factor in development of

ligamentous posttraumatic disease, comprising 11% of ankle arthritis complaints. Several other authors have reported degenerative cartilage changes in patients with chronic LAI. One study found 40.3% of arthroscopy patients to have osteochondral lesions after 7 months (Takao et al. 2005), another study noted a 26% prevalence of chondral injuries (Kibler 1996), and a third study observed a 52% rate of degenerative changes after 5 years with LAI (Ferkel and Chams 2007). When documented, the majority of these lesions are reported to occur on the anterior aspect of the medial malleolous and opposing talar dome (Hirose et al. 2004, Kibler 1996, Takao et al. 2005, Van Dijk CN 1996). For example, one study describes 84% of cartilage lesions found on arthroscopy to be in the anteromedial talocrural joint (Sugimoto et al. 2009), and another found 79% of lesions on the anteromedial part of the joint in cadaveric ankles with known LAI (Hirose et al. 2004). Harrington evaluated 36 patients with a 10 year history of recurrent ankle sprains and found evidence of degenerative arthritis in 78% (Harrington 1979), with the vast majority of lesions on the anterior or medial part of the joint. The anteromedial shift in peak cartilage contact strain observed in our study is consistent with the location of cartilage degeneration observed clinically in similar patient populations.

Although the link between LAI and arthritic changes has been extensively studied, it is difficult to define a causal relationship between the two (DiGiovanni and Brodsky 2006, Harrington 1979, Povacz et al. 1998). Taga et al. (1993) demonstrated cartilage defects in patients with LAI, and determined the lesions worsened with increased time elapsed from injury. Another study corroborated these data by comparing MR images of patients with acute ankle sprains to the results of patients with symptomatic chronic ankle sprains (Cardone et al. 1993, Frey et al. 1996). Despite reporting a greater severity of sprains, the acute patients demonstrated no osteochondral lesions compared with a 43% incidence in the chronic group (Cardone et al. 1993, Frey et al. 1996). These data might suggest that altered mechanics play a role in the degenerative changes observed after LAI. The present study excluded patients with evidence of previous chondral injury in an effort to isolate the contribution of kinematics on contact strains. Our data suggest that altered kinematics associated with chronic LAI may cause an increased peak cartilage contact strain in the anteromedial aspect of the joint, aggravating cartilage damage incurred during the initial injury. It is possible, therefore, that these altered contact strains could contribute to cartilage degeneration and the development of ankle osteoarthritis in this patient population.

The cartilage strain data from this study may have clinical implications for the management of LAI. Understanding the altered strain patterns after chronic injury suggests that surgical intervention could be beneficial in preventing progression to arthritis if strain patterns can be restored with anatomic reconstruction. More than 70 surgical procedures have been described, but it is unclear which is the most effective at preventing osteoarthritis (Bell et al. 2006, Chrisman and Snook 1969, Colville and Grondel 1995, DiGiovanni and Brodsky 2006, Evans 1953, Eyring and Guthrie 1986, Gould et al. 1980, Hamilton et al. 1993, Hennrikus et al. 1996, Krips et al. 2002, Krips et al. 2001, Schmidt et al. 2004). Restoring anatomic ankle kinematics may decrease osteoarthritic progression in patients with LAI.

There were several limitations to our study. Our study looked only at quasi-static weight bearing motion of the ankle joint and did not evaluate dynamic motion. Future studies will evaluate dynamic data collected from these individuals. In addition, due to the pain and instability experienced by these patients, we were unable to have them stand on the forceplate long enough to measure the equilibrium strains experienced in the cartilage. Instead, the time over which the intact and injured ankles were loaded was kept similar. Despite this limitation, our results consistently indicated that the strains in the injured ankle were higher and shifted anteromedially. Furthermore, this study measured one strain value throughout the thickness of the cartilage. However, cartilage strain is non-uniform, with

large variations throughout its thickness (Choi et al. 2007, Guterl et al. 2009). However, our study did quantify significant differences in the average peak strain between the injured and normal ankles and provided important insight into the differences in loading between the two sides. Computational modeling might be helpful in further characterizing the differences in loading with LAI. Lastly, we excluded patients with soft tissue injuries to structures other than the ATFL and CFL. Although the ankles of most patients with lateral instability are complicated by other problems, it was necessary for us to exclude soft tissue injury to isolate the contribution of cartilage strain due to injuries to the ATFL and CFL. Future studies might also incorporate the effects of injury on other structures within the joint.

When compared to contralateral controls, chronic LAI resulted in increased peak cartilage strain and an anteromedial shift in position of maximum strain. The location of peak strain in our study corresponds to the region of clinically-observed cartilage degeneration in the anteromedial portion of the ankle joint (DiGiovanni et al. 2000). Chronic LAI, therefore, may contribute to the development of tibiotalar joint cartilage degeneration due to altered cartilage strains.

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Figure 1.

MR images were used to create 3D models of the tibia, talus and cartilage surfaces. Strain (ϵ) across the talocrural joint was approximated from the overlap in cartilage surfaces. The penetration distance (d) was divided by the thickness of the undeformed tibial (t_1) and talar (t_2) cartilage surfaces at each point within the joint to create a strain map.

Figure 2.

A coordinate system was established on the talus of both the intact and injured ankles simultaneously using an iterative closest point technique to register the two models. This coordinate system was used to measure the location of the peak cartilage contact strain in both the mediolateral and anteroposterior directions.

Figure 3.

A cadaveric ankle was imaged using MR in a relaxed position, and again imaged after being compressed by approximately 1mm. The cartilage contact strains digitized directly from the compressed model were compared to that predicted from the strains calculated from overlap of the surfaces. Similar strain distributions were observed from the two measurements. Peak strain was within 6% and the position of the peak strain was within 0.9mm.

Figure 4.

Contact strain maps were calculated across the talocrural joint in the injured and intact ankles in each patient. Peak strain is higher in magnitude and demonstrates an anteromedial shift in the injured ankle compared with the intact ankle, as demonstrated in one patient.

Figure 5.

The mean peak strain across all patients increased in both intact and deficient ankles with increasing weight-bearing load. Additionally, there was a statistically significant increase in peak strain in the deficient ankle compared with the intact ankle at 50%, 75%, and 100% body weight in patients with lateral ankle instability ($p < 0.05$).

Figure 6.

The mean position of the peak cartilage contact strain across all patients shifted anteriorly with increasing body weight in patients with lateral ankle instability. This anterior shift was statistically significant at 50%, 75%, and 100% ($p < 0.05$).

Figure 7.

A statistically significant medial shift in the mean position of the peak cartilage contact strain across all patients was seen at 100% body weight ($P < 0.05$).