Non-Uniform Strain Distribution in Anterolateral Capsule of Knee: Implications for Surgical Repair

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ABSTRACT: The existence of a ligamentous structure within the anterolateral capsule, which can be injured in combination with the anterior cruciate ligament, has been debated. Therefore, the purpose of this study was to determine the magnitude and direction of the strain in the anterolateral capsule in response to external loads applied to the knee. The anterolateral capsule was hypothesized to not function like a traditional ligament. A 6-degree-of-freedom robotic testing system was used to apply ten external loads to human cadaveric knees ($n = 7$) in the intact and anterior cruciate ligament (ACL) deficient states. The position of strain markers was recorded on the midsubstance of the anterolateral capsule during the resulting joint kinematics to determine the magnitude and direction of the maximum principal strain. The peak maximum principal strain ranged from 22% to 52% depending on the loading condition. When histograms of strain magnitude values were analyzed to determine strain uniformity, the mean kurtosis was 1.296 ± 0.955, lower than a typical ligament, and the mean variance was 0.015 ± 0.008, higher than a typical ligament. The mean angles of the strain direction vectors compared to the proposed ligament ranged between 38° and 130° ($p < 0.05$). The magnitude of the maximum principal strain in the anterolateral capsule is much larger than a typical ligament and does not demonstrate a uniform strain distribution. The direction of strain is also not aligned with the proposed ligament. Clinical Significance: Reconstruction methods using tendons will not produce normal joint function due to replacement of a multi-axial structure with a uni-axial structure. © 2019 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 37:1025–1032, 2019.

Keywords: strain; anterolateral capsule; knee; anterolateral ligament; biomechanics

Injuries to the anterolateral capsule are often under diagnosed in conjunction with anterior cruciate ligament (ACL) injuries, which can lead to decreased rotational stability of the knee and early arthritis.[1] Significant clinical interest exists in combined anterolateral capsule and ACL injuries, especially with regards to potential surgical treatment of the capsule at the time of ACL surgery.[1,2] In 1879, the French surgeon Paul Segond described a ligamentous structure between the lateral femur and tibia within the anterolateral capsule.[3] More recently, several authors reevaluated this area and described different anatomic origins or insertions of a possible anterolateral ligament.[4–7] A thickening of the anterolateral capsule might be the reason for these findings.[8] Lately, an expert group came to the consensus, that the anterolateral ligament is part of the capsule.[9] Its contribution to joint stability remain debatable.[10–13] Traditionally, ligaments transmit tensile loads along their length, in the same direction as their collagen fibers, and are stretched along their longitudinal axis, resulting in a relatively uniform strain pattern at their midsubstance.[14]

Knowledge of the function of the anterolateral capsule can aid in the development of an injury model and more informed choices for repair. In turn this knowledge can help to restore joint kinematics closer to its native dimensions, and to estimate the healing potential of the anterolateral capsule for the possible development of non-operative treatments options.

The purpose of this study was to determine the magnitude and direction of the strain in the anterolateral capsule in response to external loads applied to the intact and ACL-deficient knee throughout a range of flexion angles. It was hypothesized that the anterolateral capsule does not behave like a traditional ligament based on the magnitude and direction of the maximum principal strain.

METHODS

Experimental Set-Up

After ethical approval was obtained, seven fresh-frozen human cadaveric knees (mean age 53.7 years, range 46–59 years) were used in this study. Each specimen was examined manually and radiologically before testing to exclude any specimens with ligament or bony abnormalities. Specimens were thawed overnight at room temperature for 24 h before testing,[15] and the tibia and femur were cut 20 cm from the joint line. The fibula was fixed to the tibia using a bicortical screw to maintain its anatomic position. The skin and musculature was removed exposing the femoral and tibial shaft leaving the knee joint intact. During the experimental protocol, the specimen was kept moist with saline.[15] To enable visualization of the anterolateral structures the iliotibial band was carefully dissected from the underlying tissue starting proximal and ending with the transection from Gerdy's tubercle.

Twenty-four strain markers (1.58 mm diameter) were attached to the mid-substance of the anterolateral capsule in

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a 4 × 6 grid using cyanoacrylate (Fig. 1). The markers were fixed 1 cm superior to Gerdy's tubercle and the LCL insertion and 5 mm anterior to the LCL origin and insertion. The specimens were then mounted into a robotic testing system (MJT Model FRS2010, Chino, Japan). The femur was rigidly fixed relative to the lower plate of the robotic testing system and the tibia was attached to the upper end plate of the robotic manipulator through a 6-degree-of-freedom universal force/moment sensor (UFS, ATI Delta IP60 (SI-660-60), Apex, NC). A custom digital motion capture system (Spica Technology Corporation, Haiku, HI; 0.01 mm accuracy) was used to track the 3D motion of the strain markers attached to the surface of the capsule.[16,17] Four cameras were set up surrounding the robotic testing system such that a minimum of two cameras could always capture all of the markers on the anterolateral capsule. The surface of the midsubstance of the anterolateral capsule was broken into 15 elements based on the placement of the strain markers to allow for comparisons of strain with the location of the proposed anterolateral ligament. Before application of external loads to each specimen, the reference strain configuration was obtained. The position of 30° knee flexion was determined during preliminary testing during the path of knee flexion and extension as the position in which the anterolateral capsule was almost free of wrinkles or folds. The capsule was inflated with 1 kPa of air pressure at 30° knee flexion with neutral rotation to remove any folds or wrinkles in the tissue. The reference strain configuration was determined using a modification of the procedure described by Malicky et al.[18] and has been used with the glenohumeral capsule in previous studies.[19,20]

Magnitude of Maximum Principal Strain

Only the most important loading conditions based on preliminary testing have been used for determination of tissue strains. During preliminary studies we narrowed the field to those that produced the greatest strain in the tissue. Ten loading conditions were applied to the knee and, again, the position of the markers was measured: 134 N anterior tibial load, 134 N anterior tibial load + 7 Nm internal rotation torque, 7 Nm external rotation torque, 7 Nm external rotation torque + 7 Nm varus torque, 7 Nm internal rotation torque, 7 Nm internal rotation torque + 7 Nm valgus torque, 134 N posterior tibial load, 134 N posterior tibial load + 7 Nm external rotation torque, 7 Nm valgus torque, and 7 Nm varus torque. The 10 loading conditions were applied at 30°, 60°, and 90° of knee flexion to the intact and ACL-deficient knees. The location of the strain markers for each loading configuration was input into ABAQUS (ABAQUS/CAE Student Version 6.4; Simulia, Providence, RI) as a set of 24 nodes comprising 15 elements to calculate the magnitude of the maximum principal strain. The grids were normalized such that elements on right and left knees refer to the same anatomical positions. Calculations were performed for each element at the maximum load for each loading condition as the difference in marker location between the strained configuration and the reference configuration.[16] Green-Lagrange strain was calculated in 3D assuming large deformations and non-linear geometry. The magnitude of the maximum principal strain was output for the centroid of each element.

Direction of Maximum Principal Strain

The two single loading conditions which caused the highest strain in the anterolateral capsule (anterior tibial load in the ACL-deficient knee and internal rotation torque in the ACL-deficient knee) and the loading conditions not expected to significantly deform the anterolateral capsule (posterior tibial load in the ACL-deficient knee and external rotation

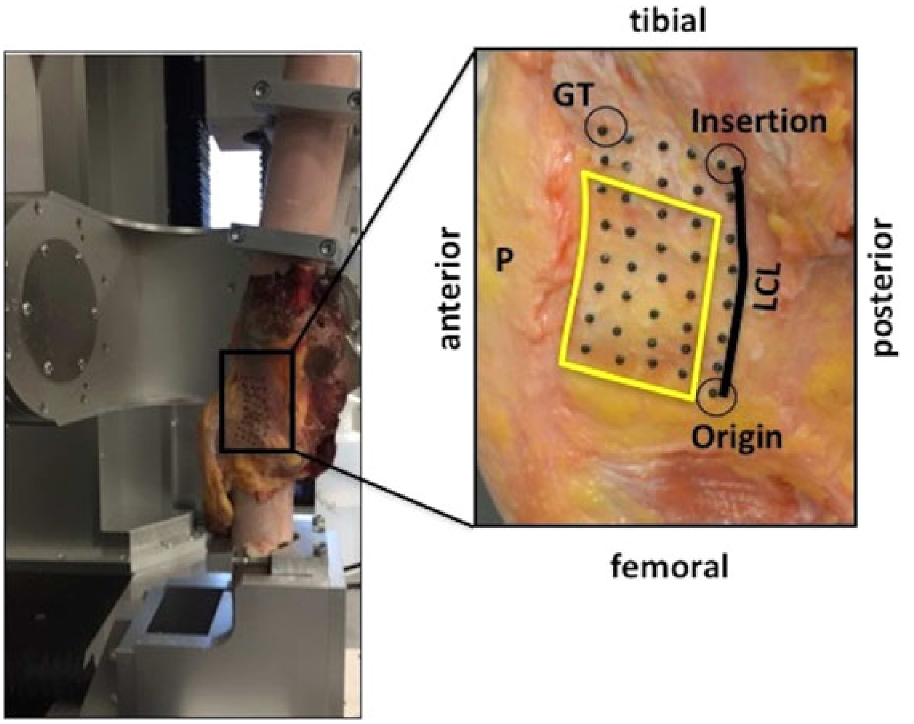

Figure 1. The knee was mounted into a robotic testing system. Twenty-four strain markers were attached to the mid-substance of the anterolateral capsule in a 4 × 6 grid using cyanoacrylate. Markers in yellow box used for analysis. GT, Gerdy's tubercle; P, patella; LCL, lateral collateral ligament.

torque in the ACL-deficient knee) were used for further analysis. Thus, the direction of the maximum principal strain for those four loading conditions at three flexion angles were determined for the ACL-deficient knee. The loads were: 134 N anterior tibial load, 7 Nm external rotation torque, 7 Nm internal rotation torque, and 134 N posterior tibial load at 30°, 60°, and 90° of knee flexion, respectively. The direction was output at each of the four element integration points. The angle between the direction vectors representing each element in the anterolateral capsule and the proposed anterolateral ligament[4] was determined in a counterclockwise manner from the proposed anterolateral ligament using Image J software (National Institute of Health, Bethesda, MD) (Fig. 2).

Statistical Analysis

A one-tailed Student's t-test was used to compare the peak maximum principal strain from across the entire anterolateral capsule of the intact and ACL-deficient knee after application of the respective loading conditions at each knee flexion angle, since the authors considered the strain in the anterolateral capsule to be higher after dissection of the ACL than with intact ACL. A two-tailed t-test with a Bonferroni correction ($p < 0.0167$) was used to compare the peak maximum principal strain at the different flexion angles.

For the four loading conditions used for further analysis, histograms of the magnitude of the strain distribution within the anterolateral capsule at each flexion angle and loading condition were generated for each specimen. The magnitudes of the maximum principal strain were compared between flexion angles and loading conditions as histograms normalized with bins from 0 to 90% strain at increments of 5%. The histograms could then be compared with a two-tailed unpaired t-test to the uniform strain distribution expected for a typical ligament based on the variance and kurtosis. The average angles for each element between the direction vectors representing each element in the anterolateral capsule and the proposed anterolateral ligament were determined per loading condition and flexion angle for the seven specimens. Angles were in the range of $0 - 180°$.[19] A one-tailed paired t-test was used to compare the angles with 0°. Significance set at $p < 0.05$.

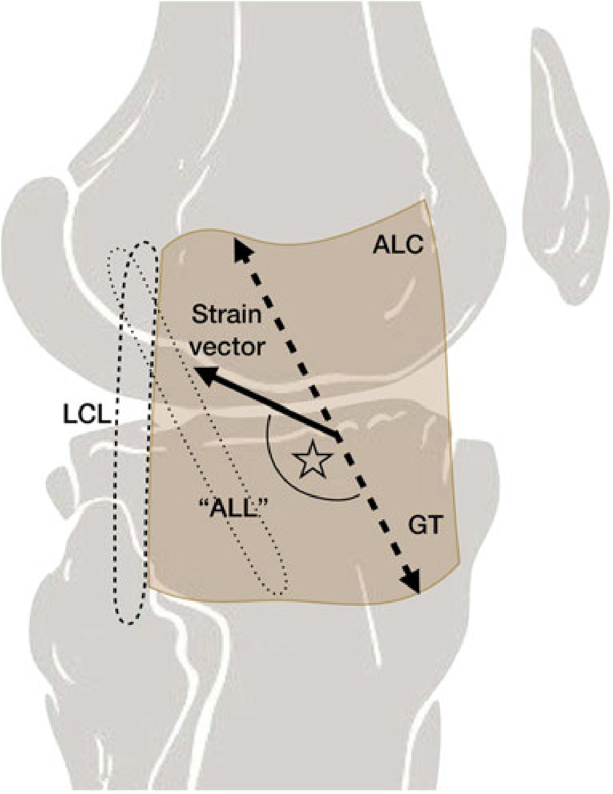

Figure 2. The angle (black star) between the direction vectors representing each element in the anterolateral capsule and the anterolateral ligament was determined in a counterclockwise manner from the proposed anterolateral ligament. LCL, lateral collateral ligament; "ALL", anterolateral ligament; ALC, anterolateral capsule. GT, Gerdy's tubercle.

RESULTS

Magnitude of Maximum Principal Strain

The peak maximum principal strain ranged from 22 to 52% depending on the loading condition and knee flexion angle. The magnitude of the peak maximum principal strain in the anterolateral capsule midsubstance was not consistent between specimens in the same loaded configurations. A fringe plot for a knee in response to external rotation torque at 90° of knee flexion with the location of the proposed anterolateral ligament highlighted by a black line is shown in Figure 3. The magnitudes of the maximum principal strain were not uniformly distributed. For this specimen, the higher strains were primarily concentrated on both the femoral and tibial sides of the anterolateral capsule anterior to the proposed anterolateral ligament. Additionally, a region of tissue, where high strain was present was in the anterior part of the anterolateral capsule. Minimal strain was observed in the midpart of the anterolateral capsule.

The highest peak maximum principal strain of the anterolateral capsule was found in ACL-deficient knees due to five loading conditions: anterior tibial load, anterior tibial load + internal rotation torque, internal rotation torque, internal rotation torque + valgus torque, and valgus torque at 30°, 60°, and 90° of knee flexion, respectively. In the ACL-deficient knee at 30° of knee flexion, the peak maximum principal strain due to these five loading conditions was significantly higher than all other loading conditions tested ($p < 0.05$). Those loading conditions excluding valgus torque had magnitudes of maximum principal strain $\geq 50\%$ (Fig. 4).

The peak maximum principal strain in response to the five loading conditions increased with knee flexion angle in the intact as well as in the ACL-deficient knee ($p < 0.0167$) (Fig. 5). During anterior tibial load in the ACL-deficient knee, the change of knee flexion from 30° to 60° increases the peak maximum principal strain by 5.9%. The change from 30° to 90° increases the peak maximum principal strain by 6.6%.

The ACL-deficient knee always had higher peak maximum principal strain than the intact knee. When comparing the ACL-deficient and intact knee states there was a significant difference ($p < 0.05$) between 14 of the 30 compared loading conditions. In response to anterior tibial load/internal rotation torque in 30° of knee flexion, the peak maximum principal strain in the ACL-deficient knee was 16.8% higher than in the intact knee.

The histograms for magnitude of the maximum principal strain did not represent normal distributions and varied between specimen, loading condition, and flexion angle. Overall, the mean kurtosis was 1.296 ± 0.955 and the mean variance was 0.015 ± 0.008 (Fig. 6). The variance is linked to the spread of the strain magnitudes between bins and the kurtosis indicates the prevalence of strain magnitudes over the range of bins. The variance of the magnitudes of the maximum principal strain in the anterolateral capsule midsubstance was found to be considerably different than the variance of the magnitude of the maximum principal strain in a typical ligament.

Direction of Maximum Principal Strain

The direction of the maximum principal strain varied based on loading condition and knee flexion angle. The average angles calculated between the direction vectors of the maximum principal strain and the proposed ligament's direction vary depending on loading condition and knee flexion angle as well (Fig. 7). The direction of the peak maximum principal strain in the anterolateral capsule midsubstance was not consistent between specimens in the same loaded configurations.

The difference between the proposed ligament and the direction of the maximum principal strain over all flexion angles for anterior tibial load was 53.6° ± 25.9°, for posterior tibial load was 69.6° ± 40.8°, for external rotation torque was 121.6° ± 23.4°, and for internal rotation torque was 43.5° ± 24.9°. The mean angles ranged between 38° and 129.9°. All angles were significantly different than 0° ($p < 0.05$).

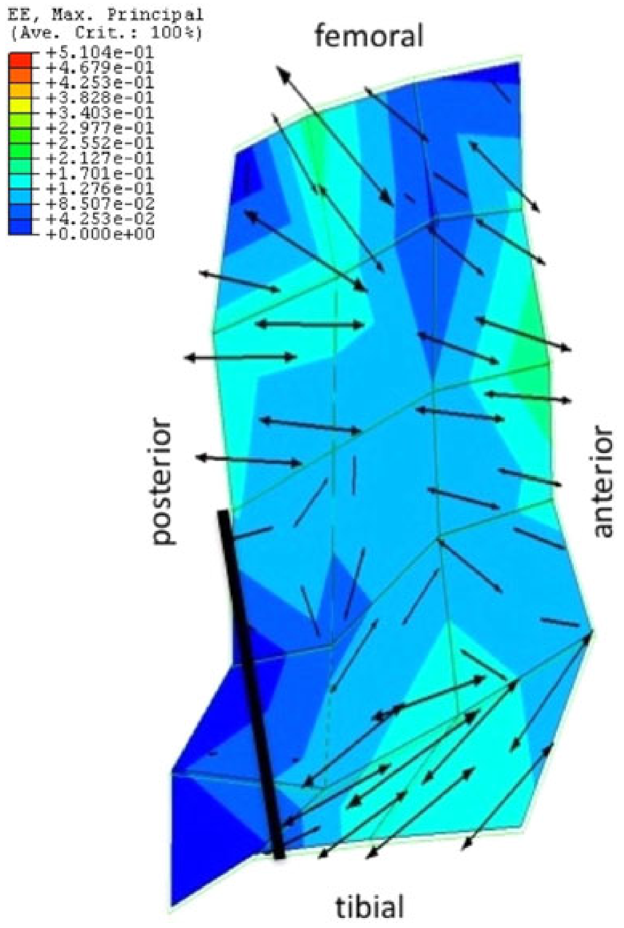

Figure 3. Sample fringe plot in response to external rotation torque at 90° of knee flexion. The green regions represent areas with higher maximum principal strain magnitudes. The black arrows represent the vectors of the maximum principal strain. The magnitudes of the maximum principal strain were not uniformly distributed. The direction vectors, **indicated by the black arrows**, varied throughout the entire anterolateral capsular midsubstance and were not aligned with the proposed ligament (thick black line).

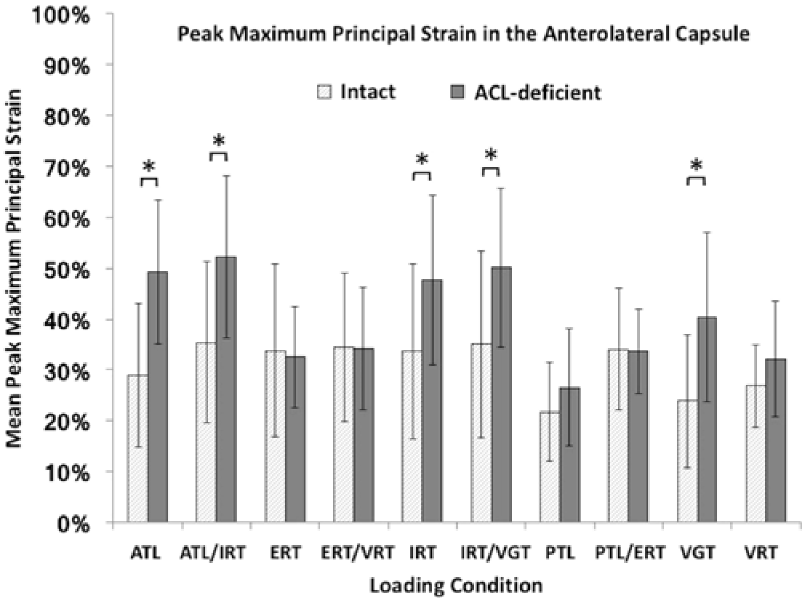

Figure 4. Peak maximum principal strain in the anterolateral capsule with the knee in 30° of flexion. ATL, anterior tibial load; IRT, internal rotation torque; ERT, external rotation torque; VRT, Varus torque; VGT, Valgus torque. PTL, posterior tibial load. (${}^{*}p < 0.05$)

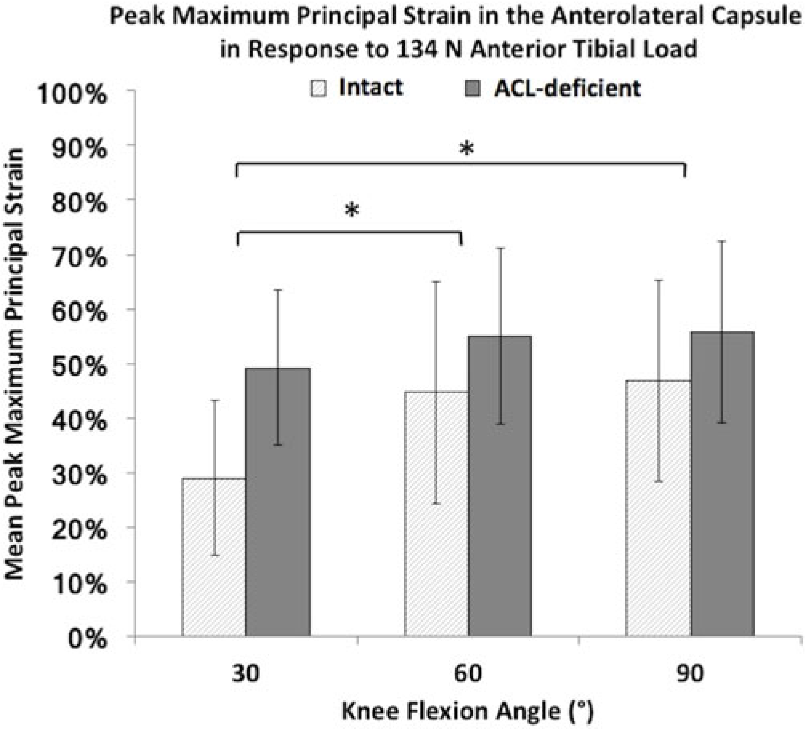

Figure 5. Peak maximum principal strain in the anterolateral capsule in response to 134 N anterior tibial load (*$p < 0.0167$).

DISCUSSION

The results from this study show that the anterolateral capsule of the knee does not deform like "traditional" ligaments and that the direction of the strain does not align with the proposed anterolateral ligament, supporting the hypothesis of the study. However, this study also shows that, even though the anterolateral capsule does not behave like a traditional ligament, it becomes an important restraint in the ACL-deficient knee, especially in higher knee flexion angles. It is known from a previous study, that the anterolateral capsule experiences high in-situ forces in the ACL-deficient knee at higher knee flexion angles.[21] In this study, the highest strain in the anterolateral capsule occurs in the ACL-deficient knee during loading conditions (anterior tibial load, anterior tibial load + internal rotation torque, internal rotation torque, internal rotation torque + valgus torque, and valgus torque) in which the ACL is known to be a primary or secondary restraint.[22–24] In the case of ACL-deficiency, higher forces are conducted to the anterolateral capsule in response to those loading conditions, leading to higher strain magnitudes in the anterolateral capsule. This is especially true for higher knee flexion angles since the magnitude of maximum principal strain increased with knee flexion angle.

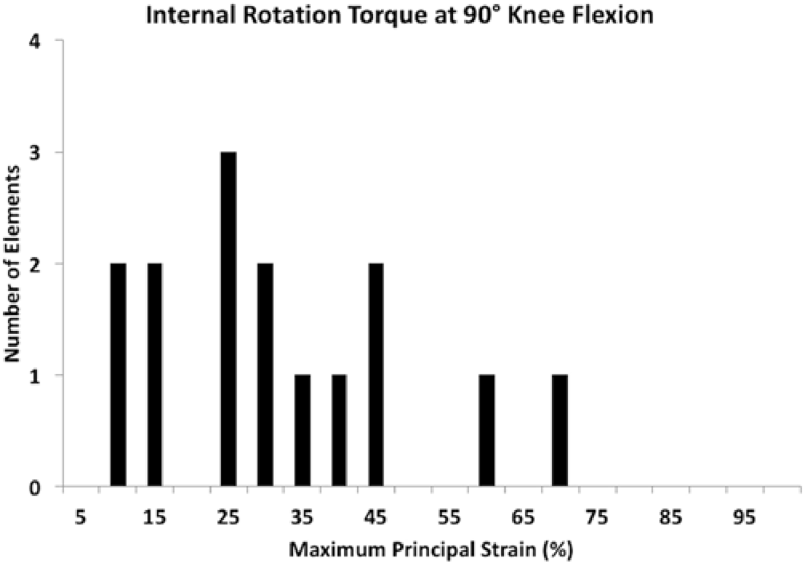

Figure 6. Histogram of magnitude of maximum principal strain normalized with elements from 0 to 90% strain at increments of 5% in response to internal rotation torque at 90° of knee flexion for typical specimen. One element was defined as the space bounded by four markers.

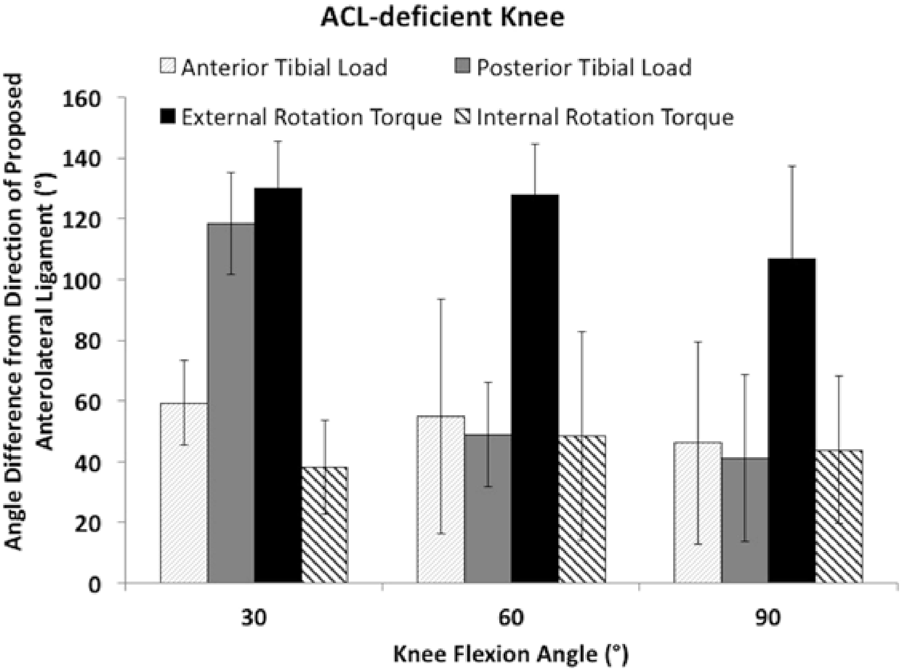

Figure 7. Angle between the maximum principal strain vectors and the proposed anterolateral ligament in the anterior cruciate ligament (ACL)-deficient knee.

Prior to conduction of the study a comprehensive preliminary study was performed to determine the joint position for appropriately defining the reference strain configuration (tissue is uniformly preloaded). The flexion and internal/external rotation angles that minimized the wrinkles and folds in the capsule when inflated with minimal pressure was 30° of knee flexion at neutral rotation. Other flexion angles had significantly greater numbers of wrinkles and folds in the capsule leading to erroneous strain predictions.

The magnitude of maximum principal strain in the anterolateral capsule was much greater than the ultimate strains of tendons and ligaments[25] (approximately doubled). In other words, the stiffness of the capsule is lower than typical tendons and ligaments at the knee. These findings suggest that a proposed ligamentous structure in the anterolateral capsule does not have tensile properties like ligaments. This is in accordance with a histological study, which compared the anterolateral capsule to the ACL.[26] The collagen pattern of the thickenings in the anterolateral capsule were organized into individual bundles, indicating that the anterolateral capsule includes a combination of multiple thickenings and not a homogenous entity such as the ACL. Another study determined structural properties, including ultimate load, ultimate elongation, and stiffness of the anterolateral capsule and the iliotibial band suggesting that the anterolateral capsule does not have structural properties like a traditional ligament.[27] The iliotibial band had almost 50% higher ultimate load and nearly three times higher stiffness compared with the anterolateral capsule. The anterolateral capsule had about double the ultimate elongation compared with the iliotibial band. Furthermore, the in situ forces of the anterolateral structures were evaluated during application of external loading conditions and the anterolateral capsule carried negligible forces in the longitudinal direction, suggesting that the anterolateral capsule does not function as a traditional ligament.[21]

The magnitudes of the maximum principal strain were not uniformly distributed nor were they distributed homogeneously. If a ligament were elongated, the magnitude of the maximum principal strain would be distributed homogeneously throughout the midsubstance, which was not the case with the anterolateral capsule over the tested loading conditions. A histogram of the magnitude of the maximum principal strain of a traditional ligament would have a high kurtosis and the variance would be low. This concept is based on the fact that ligaments have similar tissue properties throughout their midsubstance. The anterolateral capsule has variable thickness leading to different tissue properties in different regions of the capsule. Compared to expected values for a typical ligament, the anterolateral capsule kurtosis was lower than expected and the variance was higher. The variances were found to be considerably different between the anterolateral capsule and an ideal ligament.

The angles between the direction vectors of the maximum principal strain across specimens and loading conditions and the direction of the proposed anterolateral ligament were significantly different from 0°. The direction vectors varied throughout the entire capsular mid-substance. For a typical ligament, the distribution of the direction vectors would have been consistently close to the longitudinal direction of the proposed anterolateral ligament and had little variation. The human MCL has mechanical properties that are 30 times higher along its longitudinal direction compared to its transverse direction.[28]

In summary, based on the results of this study the anterolateral capsule does not function like a traditional ligament. Therefore, reconstruction methods utilizing tendon grafts will not produce normal joint function due to replacement of a multi-axial structure with a uni-axial structure. This is in accordance to a recent study showing that reconstruction of the anterolateral ligament in conjunction with an ACL-reconstruction significantly reduced rotatory laxity of the knee beyond 30° of knee flexion and resulted in significant overconstraint of the knee regardless of fixation angle.[29] Furthermore, it was shown that the impact on joint kinematics by reconstruction methods utilizing the iliotibial band like the deep Lemaire and MacIntosh highly depend on tension of the graft.[30] Further treatment options include utilization of highly compliant sheets of tissue or conservative treatment. The healing potential of the anterolateral capsule should be considered before surgical procedures are conducted.

The limitations of this study include mapping the three-dimensional direction vectors into two-dimensional space to calculate the angles with the proposed anterolateral ligament. This process introduced some variability since no two fringe plots could be oriented exactly the same to display both the magnitude and the direction of the maximum principal strain. However, when comparing the proposed anterolateral ligament to the direction vectors, the proposed anterolateral ligament was always considered to be between the same anatomical points regardless of their orientation. Furthermore, in vivo the iliotibial band is attached to the anterolateral capsule possibly altering tissue properties. Studies have shown that the iliotibial band adds rotational stability to the ACL-deficient knee.[13] However, this function is less impressive if the ACL is intact.[31] To determine the strain in the anterolateral capsule the iliotibial band had to be removed.

In the future, studies could aim at establishing injury models of the anterolateral capsule to better understand the complex injury pattern leading potentially to knee instability. This can be directly transferred to clinic, since knowledge of the behavior of the capsule can help to improve injury prevention models. Treatment options should be adapted for properties of the anterolateral capsular tissue to ensure anatomic repair or reconstruction and improve patient' outcome. However, the need for anatomical reconstruction of the anterolateral capsule may be limited in a normal-functioning ACL-reconstructed knee, since the anterolateral capsule engages only during pathologic ranges of tibial translation.[32]

CONCLUSIONS

The magnitude and direction of the maximum principal strain in the anterolateral capsule does not have a uniform strain distribution like a traditional ligament. The findings of this study lead to the assumption that reconstruction methods using tendons grafts will not produce normal joint function due to replacement of a multi-axial structure with a uni-axial structure. However, because of its non-uniform strain distribution and function in higher knee flexion angles surgeons might consider the anterolateral capsule for treatment of the injured ACL in patients.

AUTHORS' CONTRIBUTIONS

DG, SLS, KMB, SI, FHF, VM, and RD designed the research, performed acquisition, analysis, and interpretation of data. DG, VM, and RED drafted the paper and revised it critically. All authors approved the submitted and final version of the manuscript.

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