THE EFFECT OF ANTERIOR CRUCIATE LIGAMENT INJURY ON THE IN VIVO ELONGATION OF THE MEDIAL AND LATERAL COLLATERAL LIGAMENTS

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INTRODUCTION

Although anterior cruciate ligament (ACL) injury commonly leads to joint degeneration [1], few quantitative data have been reported on its effect on soft tissue structures surrounding the knee joint. In the present study, we hypothesized that ACL injury will alter the deformation of both collateral ligaments during an in vivo functional activity. We used magnetic resonance (MR) and dual-orthogonal fluoroscopic imaging techniques [2] to analyze the effects of injury to the ACL on the length of the collateral ligaments during in vivo knee flexion from 0° to 90°, with the healthy contralateral knee of each patient serving as a control.

METHODS

Six patients (five males and one female; age range 19-38 years old) with acute ACL injury in one knee and the contralateral side intact participated in this study. The protocol was approved by the Institutional Review Board at our institute. Both knees were imaged with a MR scanner to create 3D models of the knees. Each anatomic knee model included the geometry of the femur and tibia, as well as the attachment sites of the superficial medial collateral ligament (SMCL), deep medial collateral ligament (DMCL), and lateral collateral ligament (LCL) (Fig 1).

Next, both knees of each subject were imaged using two orthogonally placed fluoroscopes as the patient performed a single-leg lunge at 0°, 15°, 30°, 60°, and 90° of flexion. The models were manually manipulated in 6 degrees-of-freedom until the projections of the models matched the outlines of the fluoroscopic images. A series of knee models that reproduced knee positions at all target flexion angles recreated the in vivo knee flexion from 0° to 90° of flexion (Fig 2).

At each flexion angle, the Wilcoxon signed rank test was used to compare the length of the fiber bundles between the intact and ACL injured knees. Statistical significance was set at p< 0.05.

RESULTS

ACL injury caused a significant elongation of the fiber bundles of the SMCL at every flexion angle (p< 0.05). The fiber bundles of the SMCL lengthened around 1.5% compared to the healthy knee. Similar increases in length were observed in the DMCL bundles after ACL injury (Fig 3). The effect of ACL injury was maximal at 30° of flexion in the anterior bundle of the DMCL, with a 3.9% increase in length compared to the length of the same bundle in the intact knee. In contrast, the LCL fiber bundles shortened after ACL injury. The maximal difference between LCL bundles of the intact and injured knee occurred at 15°, where the length of the posterior bundle was 3.9% shorter compared to the length of the same bundle in the intact knee.

DISCUSSION

The altered deformations of the collateral ligaments, associated with the changes in tibiofemoral joint kinematics after ACL injury, demonstrate that deficiency of one of the knee joint structures upsets the in vivo knee homeostasis. We only examined the effect of ACL injury during a weightbearing single leg flexion. In the future, the effects of injury to the ACL on the elongation during other in vivo activities such as gait, stair ascent and descent, should also be investigated. Restoring normal knee kinematics after ACL reconstruction is critical to restore the normal function of the collateral ligaments.

REFERENCES


Figure 1. A typical 3D model of the knee created from sagittal plane MR images. (A) The attachment sites of the SMCL and DMCL; (B) the attachment sites of the LCL.

Figure 2. The knee models for a typical subject at 0°, 15°, 30°, 60°, and 90° of flexion during weightbearing lunge.

Figure 3. Rupture of the ACL caused a significant elongation of the fiber bundles of the DMCL at every flexion angle (* p< 0.05).