ABNORMAL AMBULATION AFTER ACL RUPTURE AFFECTS KNEE ARTICULAR CARTILAGE SPATIAL THINNING PATTERN

INTRODUCTION:
Premature osteoarthritis (OA) is frequently reported in patients with anterior cruciate ligament (ACL) deficient knees [1]. Yet there is a paucity of information for the causes of early OA in ACL deficient knees. One possible explanation for premature OA in ACL deficient knees is associated with the kinematic changes following ACL injury during ambulation [2]. Our previous report [3] has suggested that the kinematic changes after ACL rupture can initiate a pathway to knee OA. The kinematic changes in the knee following ACL injury during walking may shift the frequent contact regions on both femoral and tibial cartilage to regions of the cartilage not conditioned to altered joint loading. This observation is supported by a report that ACL deficient knees have different cartilage degeneration patterns when compared with ACL intact knees in patients with end stage knee OA [4]. However, at present there has not been any direct evidence that kinematic changes initiate the early cartilage thinning and events that lead to clinical symptoms. To address this need this study examined the possibility that early patterns of cartilage thinning in ACL deficient population, are related to changes in knee kinematics after ACL rupture. It was hypothesized that patterns of cartilage thinning are related to kinematic changes between ACL injured and uninjured knees.

METHODS:
17 unilateral ACL deficient subjects (11 males and 6 females, age 41.1±12.1 years, BMI 24.2±4.8 kg/m²) were recruited for the study. After IRB approval and informed consent were obtained, the subjects underwent a gait test at self-selected walking speed, and a MR imaging for both knees. The point cluster technique [5] was used to calculate knee kinematics including anterior-posterior (AP) translation and internal-external (IE) rotation of femur relative to tibia. Average AP translation and IE rotation during stance phase of walking were calculated for both ACL intact and deficient knees. The AP translational and IE rotational offsets were calculated by subtracting the average AP translation and IE rotation of ACL intact knee from those of ACL deficient knee, respectively for each ACL deficient subject. Knee MR images were obtained using a fat-suppressed 3D SPGR sequence. Tibial cartilage in the bilateral knee MR images were segmented and reconstructed into 3D models with thickness maps [6]. The cartilage thickness maps were projected onto planes. The projected thickness maps from contralateral ACL intact knees were subtracted from the projected thickness maps from ACL deficient knees to create difference maps (Fig. 1).

(a) Thickness Map
(b) Difference Map

The thinning volume in each region normalized by its surface area was calculated for the ACL deficient knee for each subject. For all subjects, the difference of thinning in anterior and posterior regions were tested using the paired Student’s t-test at α = 0.05. The subjects were divided into anterior (n=7) and posterior tibial contact (n=8) groups. Lines connecting the medial and lateral contact points were drawn.

RESULTS:
The amount of cartilage thinning was associated with the specific kinematic changes that caused a shift to either the posterior or anterior tibia (Fig 2). The subjects with posterior tibial contact in ACL deficient knee relative to ACL intact knee (Fig 2) had significantly larger thinning in anterior regions than posterior regions (Fig 3), and the subjects with anterior tibial contact had marginally larger thinning in posterior regions than anterior regions.

DISCUSSION:
The results support the theory that the kinematic changes in ACL deficient knee affect the spatial articular cartilage thinning pattern in the knee. Interestingly there were two different kinematic patterns that caused different contact patterns and for both cases the thinning was prevalent in region that was unloaded after ACL rupture. Previous studies have shown that unloading or reduced loading affected cartilage atrophy [7, 8]. This study implies that the subtle change in knee kinematics after knee injury may cause early cartilage thinning by changing the load distribution. These patterns of early thinning may be markers of events leading to the initiation of premature OA in patients following ACL injury. These results suggest the restoring rotation and translational kinematics after knee injury might be important to maintain healthy cartilage after knee injury.

REFERENCES:

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