Longitudinal texture changes to UTE-T2* following ACL reconstruction

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Introduction: Transchondral impaction to the posterior lateral tibial plateau (pLTP) is commonly sustained during pivot-shift anterior cruciate ligament (ACL) injury and causes both local bone "bruising" (bone-marrow contusions) and cartilage trauma[1,2]. A recent examination of cartilage injuries after ACL tear by Potter *et al* noted that the most severe injuries occurred in the lateral compartment with 85% of patients demonstrating bruising to the LTP, and 88% of patients demonstrating LTP cartilage loss, especially to the posterior margin of the lateral tibial plateau[2]. Quantitative ultrashort TE-enhanced T2* mapping (UTE-T2*) is sensitive to short T2 signals emanating from deep layers of cartilage, reflecting collagen structural integrity[3], and has shown the potential to detect sub-clinical cartilage degeneration[4]. Through image texture analyses, the spatial distributions of quantitative MRI parameters, including T2 and T1rho, have recently been shown to indicate disruptions to cartilage homeostasis[5,6]. This study tests the hypothesis that texture analysis of longitudinal UTE-T2* acquisitions will be better able to detect and quantify sub-surface cartilage damage and disease progression to pLTP cartilage in ACL-injured subjects than mean UTE-T2* alone.

Methods: Twenty-six subjects participated in these studies: 11 uninjured control subjects (5 female, 28±4 yrs; BMI 25±4) with no known or suspected knee pathology and 15 subjects with ACL injury requiring ligament reconstruction surgery (9 female, 28±9 yrs; BMI 29±7). All subjects provided informed consent for these IRB-approved studies. ACLT subjects were scanned twice: prior to (pre-SX) and 2 years after ACL reconstruction surgery; uninjured controls were scanned once. 3-D DESS (dual-echo steady state) images, acquired on the ACL-injured subjects with the parameters suggested by the NIH-sponsored Osteoarthritis Initiative program[7] , were read for presence of bone bruise. 3-D AWSOS (acquisition-weighted stack of spirals)[8] images were acquired on all subjects on a 3T Siemens Tim Trio scanner with an 8-channel knee coil. Eleven images at TE= 0.6-40ms were collected with FOV=140mm, matrix size=256, and in-plane resolution=0.55mm at 2mm slice thickness. Other acquisition parameters were: 60 slices, 24 in-plane spirals, 11.52ms spiral readout time, 5µs data sampling interval, and FA/TR 30°/80ms. Scan time was 1.92 min per TE-image. These TE-images were linearly interpolated to a matrix size of 512 prior to T2*-curve fitting. UTE-T2* maps were generated with a mono-exponential pixel-by-pixel T2-fit routine using MRIMapper software (© Beth Israel Deaconess and MIT 2006). The pLTP cartilage was manually segmented from a lateral section; full-thickness regions of interest (ROIs) were further segmented to separately evaluate the superficial and deep portions of the pLTP cartilage. UTE-T2* map texture features were assessed in full-thickness ROIs with grey-level co-occurrence matrix (GLCM) image analysis using in-house developed software written in MATLAB (The MathWorks, Natick, MA) based on the 'graycomatrix' and 'graycoprops' functions. Four statistical features ('contrast', 'homogeneity', 'correlation' and 'energy') were investigated at three different spacings (1, 3, 5 pixel offsets) in 2 directions, parallel and perpendicular to cartilage laminae. The distributions of UTE-T2* mean values and GLCM statistics were assessed for normality using Shapiro Wilks tests. Two-tailed t-tests assessed differences between ACL-injured and uninjured control's UTE-T2* and GLCM statistics. Paired two-tailed t-tests evaluated longitudinal changes in UTE-T2* and GLCM metrics. In cases where the data was not found to meet the assumptions of a normal distribution, non-parametric Mann-Whitney U Tests (MWUT), and paired Wilcoxin Signed Ranks Tests were used. Pearson correlations assessed the relationship between change in GLCM and change in UTE-T2* means. All statistical analyses were performed with IBM SPSS.

Results: Mean UTE-T2* values in full-thickness, superficial and deep articular cartilage ROIs of the pLTP of ACL-injured subjects (n=15) were not found to discriminate ACL-injured subjects (n=15) from uninjured controls (n=11) either pre-SX or 2 years after ligament reconstruction (P>0.19). Texture analysis of UTE-T2* maps, however, did differentiate ACL-injured subjects from uninjured controls in 13 of 24 texture features tested pre-SX and in 15 of 24 textures features tested 2 years post-SX. Specifically, the distribution of pLTP UTE-T2* values in ACL-injured subjects, evaluated perpendicular to cartilage laminae, was significantly less contrasty, more homogeneous and more correlated than in uninjured controls at both time-points (P<0.05). Measured in the direction parallel to cartilage laminae, the distribution of UTE-T2* values in ACL-injured subjects was more significantly more contrasty, less homogeneous and less correlated than in uninjured controls at both time-points (P<0.05). Only the energy statistic did not differentiate between ACL-injured and uninjured control cartilage in any direction, spacing or time-point tested (P>0.05). Longitudinally, 3 of 24 texture statistics changed significantly between pre-SX and 2-year evaluations in ACL-injured subjects. The correlation statistic, measured parallel to cartilage laminae decreased overtime at both 3 and 5 pixel offsets (P=0.3, 0.3), while homogeneity measured perpendicular to the cartilage laminae, 5 pixel offset, increased significantly following ACL reconstruction (P=0.05). Longitudinally, mean UTE-T2* values in full-thickness, superficial and deep pLTP ROIs were not found to change significantly between pre-SX and 2-year evaluations in ACL-injured subjects (P=0.67, 0.23, 0.09). None of the pLTP cartilage texture statistics nor mean UTE-T2* values of ACL-injured subjects was found to vary with the presence of local bone bruise (P>0.05) or tear to the lateral meniscus (P>0.02) at either time-point. Neither 2-year change in any texture statistics nor 2-year change in mean UTE-T2* value varied with presence of bone bruise (P>0.32) or lateral meniscus tear (P>0.4), except for mean UTE-T2* in superficial pLTP cartilage that decreased significantly in knees with torn lateral menisci (P=0.03). Two-year change in the texture statistics did not correlate to 2-year change in mean UTE-T2* value in either bulk, superficial or deep pLTP cartilage (P>0.24).

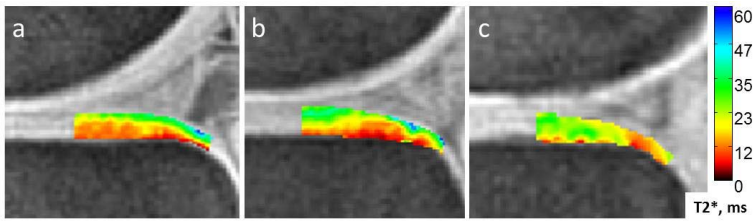

Figure 1. Sample pLTP UTE-T2* maps from (a) an uninjured control, (b) a subject with torn ACL and torn lateral meniscus prior to ACL-reconstruction, (c) and the same subject 2 years after surgery. Texture analyses discriminated uninjured controls from ACL-injured subjects at both time-points (P<0.05). In ACL-injured subjects, significant longitudinal changes to the spatial distribution of UTE-T2* values in pLTP cartilage were reflected in GLCM texture statistics that were not reflected in changes to the mean UTE-T2* values over the same time period.

Conclusions: Transchondral impaction injury to the posterolateral tibial plateau is common in the acute ACL-injured knee as evidenced by bone bruising and degeneration in the overlying cartilage. In the defined pLTP ROIs studied here, longitudinal image texture evaluation detected significant differences in the spatial distributions of UTE-T2* values within and between cartilage laminae of ACL-injured subjects compared to uninjured controls that were not reflected in the mean UTE-T2* values in the same ROIs. Furthermore, while neither superficial nor deep mean pLTP UTE-T2* values changed significantly in ACL-injured subjects over 2 years following ligament reconstruction, GLCM texture statistics in these regions showed a significant loss of correlation and increase in homogeneity over 2 years, consistent with progressive degeneration[9,10]. Assessment of changes to the spatial distribution of UTE-T2* values in cartilage augments the ability of UTE-T2* mapping to quantitatively monitor cartilage status in knees at risk of developing OA.

References: **[1** Sanders, RadGraph, 2000;20:S135**.** **[2]** Potter, AJSM, 2012;40:276. **[3]** Williams, OACart, 2010;18(4):539 **[4]** Chu AJSM in press **[5]** Baum, ArthCarRes, 2013;65(1):23. **[6]** Li, MRM, 2009;61:1310. **[7]** www.oai.ucsf.edu **[8]** Qian, MRM, 2008;60(1):135. **[9]** Carballido-Gamio, MRM, 2011;65(4):1184. **[10]** Joseph, ArthResTher, 2011;13:R153. **Acknowledgments:** NIH funded, RO1 AR052784 (CR Chu).