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INFRA-PATELLAR FAT PAD MORPHOLOGY AND MRI SIGNAL DISTRIBUTION IN ADVANCED RADIOGRAPHIC KNEE OA – DATA FROM THE OAI

F. Eckstein †‡, J. Diepold †, A. Ruhdorfer †, T. Dannhauer †‡, W. Wirth †‡, A. Guermazi §‖. † *Paracelsus Med. Univ., Salzburg & Nuremberg, Austria;* ‡ *Chondrometrics GmbH, Ainring, Germany;* § *Boston Univ. Sch. of Med., Boston, MA, USA;* ‖ *Boston Imaging Core Lab, Boston, MA, USA*

Purpose: Recent studies suggest that obesity-related risk of knee OA is not fully explained by mechanical challenge, i.e. increased body weight. Yet, adipokines (e.g. leptin) secreted from fat tissue appear to cause low-grade inflammation, and to contribute to the release of metallo-proteinases and growth factors, which are known to promote cartilage loss and osteophyte formation. The infra-patellar fat pad (IPFP) is unique in representing intra-articular adipose tissue, and is a source of intra-articular leptin. Only few studies, however, have studied the role of the IPFP in human knee OA. Here we explore whether quantitative imaging measures of IPFP size and MRI signal differ between painful knees with and without advanced radiographic knee OA in the same person, and between OA and healthy reference knees.

Methods: The sample was drawn from the Osteoarthritis Initiative (OAI) cohort and fulfilled the following criteria: OARSI medial JSN in one knee, no-JSN in the contra-lateral knee, BMI>25, frequent knee pain in both knees. The 28 participants who displayed OARSI JSN grades 2 or 3 in one (and grade 0 in the other) knee were matched 1:1 with subjects from the OAI healthy reference cohort without knee pain radiographic signs, or risk factors of incident knee OA, based on the same sex and similar body height. A sagittal fat-suppressed IM-weighted TSE sequence was used for segmentation of the entire IPFP (J.D.), volumetric 3D analysis of its morphology and signal, and for semi-quantitative evaluation of MOAKS synovitis scores (A.G.). Paired t-tests were used to compare JSN with contra-lateral non-JSN knees, and with matched reference knees.

Results: The 28 JSN participants (21 medial JSN grade 2 and 7 grade 3; 21 women, 7 men) were 165±11 cm tall (age 61±10y; weight 87±14 kg, BMI 32.2±4.4). The matched healthy reference subjects were of similar height (165±10cm), but younger (54±6y) and less heavy (70±13 kg; BMI 26±2.7). No significant differences in IPFP volume, surface areas, or thickness were observed between JSN and no-JSN knees. Compared with healthy reference knees, JSN knees displayed a 10% larger IPFP anterior surface area ($p<0.01$), but a slightly lower thickness, and no significant difference in IPFP volume (Table 1).

JSN knees displayed a significantly greater mean MRI signal intensity (SI) and greater MRI SI heterogeneity (i.e. standard deviation [SD]) than the no-JSN knees; further JSN knees displayed a substantially and significantly greater SD SI than healthy reference knees (Table 1: (b) = $p<0.01$; (c) = $p<0.001$)).

Table 1
IPFP morphometry and MRI signal intensity (SI) in JSN/no-JSN/healthy (HR) knees.

	JSN knees	noJSN knees	HR knees	Δ% JSN/noJSN	Δ% JSN/HR
Volume (cm^3)	24.7 ± 6.38	24.3 ± 6.21	23.2 ± 6.61	+1.5%	+6.4%
Ant. Area (cm^2)	19.4 ± 4.27	19.0 ± 4.40	17.6 ± 4.27	+2.2%	+10%(b)
Thickness (mm)	12.7 ± 1.65	12.9 ± 1.92	13.2 ± 1.60	-1.3%	-3.6%
MRI SI mean	152 ± 36.4	136 ± 31.5	140 ± 27.6	+11%(b)	8.0%
MRI SI SD	78.5 ± 22.2	69.8 ± 18.1	57.5 ± 10.2	+13%(b)	+37%(c)

The mean MOAKS score for Hoffa synovitis was 1.04 in the JSN knees, 0.89 in the no JSN knees (no significant difference), and 0.19 in the healthy reference knees. When studying the relationship between the MOAKS synovitis scores and IPFP MRI SI across all 84 knees, those with a grade 2/3 synovitis score (n=9) displayed greater mean SI than those with grade 0 (n=35; ANOVA post hoc: $p<0.05$ with and without adjustment for multiple comparisons). Knees with a grade 1 synovitis score (n=40) also tended to display greater mean SI than those with grade 0 ($p<0.05$ without, but not with adjustment). Knees with a grade 2/3 score and those with grade 1 scores both displayed greater IPFP SI SDs (heterogeneity) than those with grade 0 ($p<0.05$ with and without adjustment, respectively); those with grade 2/3 also tended to display greater SDs than those with grade 1 Hoffa synovitis scores ($p<0.05$ without but not with adjustment) (Fig. 1). The findings were generally identical when excluding the most peripheral row of IPFP voxels from the MRI SI analysis.

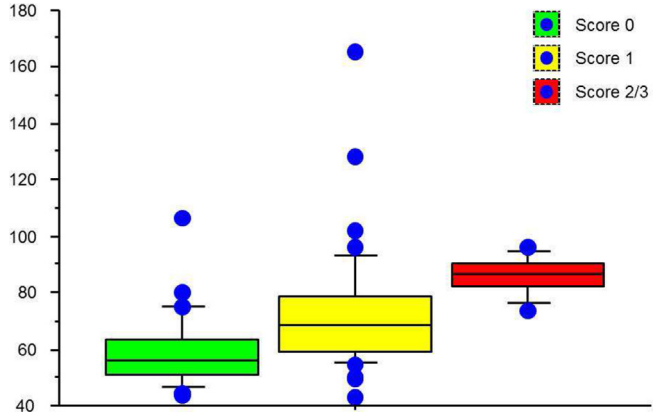

Fig. 1. Box Plot of IPFP SD MRI SI per Hoffa Synovitis Score (MOAKS).

Conclusions: This pilot study suggests that the mean intensity and heterogeneity of the MRI signal of the IPFP differs between knees with and without JSN; and the heterogeneity also between JSN and healthy reference knees. In contrast, no significant differences between groups were observed in IPFP morphology. Although IPFP MRI signal represents a relatively unspecific imaging marker, it appears to be related to semi-quantitative MOAKS scores of Hoffa synovitis.

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INCIDENT ACCELERATED KNEE OSTEOARTHRITIS IS CHARACTERIZED BY ACUTE MENISCAL PATHOLOGY WITH EXTRUSION AND ACUTE SUBCHONDRAL DAMAGE: DATA FROM THE OSTEOARTHRITIS INITIATIVE

J.B. Driban †, R.J. Ward †, C.B. Eaton ‡, G.H. Lo §‖, L.L. Price †, B. Lu ¶, T.E. McAlindon †. † *Tufts Med. Ctr., Boston, MA, USA;* ‡ *Ctr. for Primary Care and Prevention, Alpert Med. Sch. of Brown Univ., Pawtucket, RI, USA;* § *Med. Care Line and Res. Care Line, Houston Hlth.Services Res. and Dev. (HSR&D) Ctr. of Excellence Michael E. DeBakey VAMC, Houston, TX, USA;* ‖ *Section of Immunology, Allergy, and Rheumatology, Baylor Coll. of Med., Houston, TX, USA;* ¶ *Brigham & Women's Hosp. and Harvard Med. Sch., Boston, MA, USA*

Purpose: Knee osteoarthritis (KOA) is typically a slowly progressive disorder; however, a subset of knees progress with dramatic rapidity (e.g. from normal appearance to end-stage disease within 4 years). Knee injuries are a key risk factor for accelerated KOA but the existing literature is limited to self-reported injuries. We hypothesized that accelerated KOA is precipitated by an injury that destabilizes the menisci and compromises the function of a meniscus. Therefore, we conducted a preliminary but comprehensive assessment of magnetic resonance (MR) images acquired before and after self-reported injuries among individuals with accelerated KOA, common KOA, and no KOA to determine the type of acute findings associated with accelerated KOA after an injury.

Methods: We conducted longitudinal MR imaging readings among participants in the Osteoarthritis Initiative who had no radiographic KOA at baseline (Kellgren-Lawrence [KL]<2). Participants could have one of three outcomes: 1) accelerated KOA: at least one knee progressed to end-stage KOA within 48 months, 2) common KOA: at least one knee