Medial Tibiofemoral Joint Contact Force Unloading Associated with Early Knee Osteoarthritis after ACL Injury and Reconstruction

Elizabeth Wellsandt, DPT¹, Emily S. Gardinier, M.S.¹, Kurt Manal, Ph.D.¹, Michael Axe, MD², Thomas S. Buchanan, Ph.D.¹, Lynn Snyder-Mackler, PT, ScD, SCS, ATC, FAPTA³.

¹University of Delaware, Newark, DE, USA, ²University of Delaware, First State Orthopaedics, Newark, DE, USA.

Disclosures:

Introduction: Injury to the anterior cruciate ligament (ACL) is common, with over a quarter million injuries occurring annually in the United States resulting in more than 125,000 ACL reconstructive surgeries (ACLR) [1,2]. Long-term knee joint health is a growing concern, as ACL injury and reconstruction are risk factors for future knee joint osteoarthritis (OA) [3]. The exact mechanisms leading to the early development of this disease remain unclear. However, joint loading is one important factor for the development and progression of OA. Altered joint loading following ACL injury can be present despite resolution of range of motion (ROM), pain, effusion, and antalgic gait patterns [4]. Identifying a link between abnormal joint loading and the development of early knee joint OA in patients following ACL injury may be an important component in designing and implementing effective rehabilitation programs to decrease the risk of joint degeneration and enhance long-term quality of life. The purpose of this study was to determine whether knee joint contact forces during walking before or after ACLR were different in those with radiographic knee OA 5 years after surgery than in those without OA.

Methods: Fourteen patients (6 females, 8 males, mean age 32.5±12.0) with acute, unilateral ACL injury participating in cutting and pivoting activities were included in this study. All patients underwent ACLR using a hamstring autograft or allograft by a single orthopedic surgeon. Exclusion criteria included repairable meniscus injury, symptomatic grade III injury to other knee ligaments, and full-thickness chondral defects greater than 1 cm². Baseline testing was completed when impairments of ROM, effusion, and pain were resolved. Testing consisted of gait analysis with electromyography (EMG) at the following 4 time points: prior to pre-operative rehabilitation (baseline), immediately following 10 sessions of pre-operative rehabilitation (post-training), 6 months after ACLR following criterion-based rehabilitation (6 months) [5], and 2 years after ACLR (2 years). Motion analysis methods (8-camera system (Vicon), force platform (Bertec)) were used to obtain stance phase kinematics and kinetics as previously described [6] during walking at a self-selected speed. Self-selected speed was determined for each patient at baseline and was matched at follow-up testing sessions (±5%) to allow for comparison across time points.

Knee joint contact forces were calculated using an EMG-driven musculoskeletal model to estimate muscle forces [6,7]. Surface EMG of 10 muscles crossing the knee joint was input to a Hill-type muscle model which was anatomically scaled and calibrated for each subject. The medial compartment contact point was fixed at a distance of 25% tibial plateau width from the joint center. Model-derived muscle forces were used to calculate peak medial compartment contact forces (pkMC) during stance phase. Contact forces were normalized to body weight (BW) and three trials per limb were averaged for analysis.

Results: Nine subjects had OA in the index knee 5 years after ACLR, 5 did not. The OA and nonOA groups were not different with respect to BMI, sex, age, pre-injury activity level, time from injury to pre-training, or graft type. In general, the OA patients walked slower (1.58 m/s) than the nonOA patients (1.73 m/s). At 6 months the OA group had higher Marx Activity scores (nonOA: 5.0 ±3.8, OA: 9.9 ±2.5).

There was no difference in pkMC between groups at baseline (p=.209, nonOA: 2.97 BW ±0.96, OA: 2.45 BW ±0.31) (Figure 1). After pre-operative training, the OA group had significantly lower pkMC than the nonOA group (p=.032, nonOA: 3.41 BW ±1.00, OA: 2.49 BW ±0.29). Six months after ACLR (including post-operative rehabilitation) the lower pkMC persisted in the OA group (p=.038, nonOA: 3.24 BW ±0.63, OA: 2.31 BW ±0.61). Two years after ACLR, there were no longer significant differences between groups as the OA group loading increased (p=.598, nonOA: 3.06 BW ±0.60, OA: 2.94 BW ±0.16).

Discussion: Patients with radiographic medial compartment knee OA 5 years after ACLR had lower joint contact forces (unloading) in the medial compartment before and 6 months after ACLR. Patients who developed OA by 5 years had higher Marx Activity scores at 6 months than those who did not develop OA. Both groups loaded similarly 2 years after ACLR and those joint loads were not associated with the presence OA at 5 years. This persistent (months) unloading followed by reloading as
evidenced by the Marx scores and 2 year loading data may be a perfect storm for the development of knee OA after ACL injury and reconstruction.

Andriacchi and colleagues suggested that subtle changes in walking mechanics after knee injury causes early cartilage thinning [9]. They found that early cartilage thinning was more prevalent in the region that was unloaded after ACL rupture [10]. The time frame between injury and 6 months after ACLR may represent a critical period during which articular cartilage health is highly sensitive to joint unloading and cartilage deconditioning. Higher frequency of activities early after ACLR in combination with altered joint loading may together increase the risk for the development of OA.

Surprisingly, walking speed in those who developed OA was actually slower than those who did not. Although the speed was lower, both were well above average walking speed for healthy adults of 1.4 m/s.

**Significance:** These data demonstrate a potential association between early medial compartment unloading and the presence of radiographic medial knee OA 5 years later. Identifying patients with persistent altered loading and building rehabilitation programs to reduce these changes may decrease the risk for the development of OA.

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**References:**
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**Figure 1:** Peak joint contact force at the medial compartment (pkMC) during stance phase of walking. Asterisk represents p<0.05 between groups. Bars represent ±1 SD.

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