Hip Articular Loading in At-Risk and Established Hip Osteoarthritis

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Summary

Insufficient, excessive, and/or concentrated articular loading can drive osteoarthritic processes and symptom worsening. This study compared hip contact force magnitude, position, and spread during walking between at-risk and established hip osteoarthritis (OA) cohorts, and healthy controls. Only those with established hip OA had lower magnitude loading than healthy controls, which was located closer to the acetabular centre and constrained to a smaller region of cartilage.

Introduction

Suboptimal articular loading contributes to hip OA onset and progression. Insufficient, excessive, and/or concentrated articular loading can drive OA processes and symptom worsening [1]. Determining whether hip loads vary from at-risk (i.e., femoroacetabular impingement syndrome (FAIS)) to established hip OA could identify modifiable mechanisms of disease early in the OA process. This cross-sectional study compared hip contact force magnitude, position, and spread during walking between individuals with mild-to-moderate hip OA, FAIS, and healthy controls.

Methods

Nineteen participants with mild-to-moderate hip OA (age=61.3±6.4 yrs, body mass index (BMI)=29.8±4.1 kg.m^{-2}, 26% male) walked on an instrumented split-belt treadmill, 24 participants with FAIS (age=27.3±6.0 yrs, BMI=24.5±2.5 kg.m^{-2}, 79% male), and 39 healthy, pain-free controls (age=29.0±5.2 yrs, BMI=23.1±2.6 kg.m^{-2}, 62% male) walked overground at a self-selected speed while three-dimensional whole-body motion, ground reaction forces, and electromyography (EMG) from gluteus maximus, gluteus medius, medial hamstring, tensor fascia latae were synchronously recorded. Hip contact forces were calculated for 3 gait cycles per participant using an EMG-assisted neuromusculoskeletal model [2], following a synergy-based calibration [3]. Position of acetabular loading was calculated as the intersection between hip contact force and a sphere fitted to the acetabulum. Spread of acetabular loading was calculated as the great-arc distance between the instantaneous and mean positions of acetabular loading, and projected to the mean acetabular surface area. Hip contact force magnitude (bodyweights=BW), position (°), and spread (mm) were compared between groups across stance using an ANOVA *via* statistical parametric mapping (P<.05).

Results and Discussion

Participants with hip OA walked with lower magnitude hip contact forces during loading response (0-25% stance, mean difference -0.75 BW 95%CI (-0.34, -1.16) (FAIS), -0.61 BW (-0.22, -1.00) (controls), P<.01) and terminal stance (81-100% stance, -1.06 BW (-0.62, -1.50) (FAIS), -0.97 BW (-0.57, -1.38) (controls), P<.01) (Figure 1). Participants with hip OA walked with hip contact forces directed closer to the acetabular centre from mid to terminal stance (50-100% stance, -8.9° (-11.5, -6.5) (FAIS), 65-100% stance, -8.3° (-11.6, -4.9) (controls), P<.01). The spread of loading was also smaller in the hip OA group compared to FAIS (-4.3 mm (-5.9, -2.7), P<.01) and control (-4.0 mm (-5.6, -2.4), P<.01) groups, primarily during early stance (0-40%). Hip contact force magnitude, position, and spread were not significantly different between FAIS and control groups.

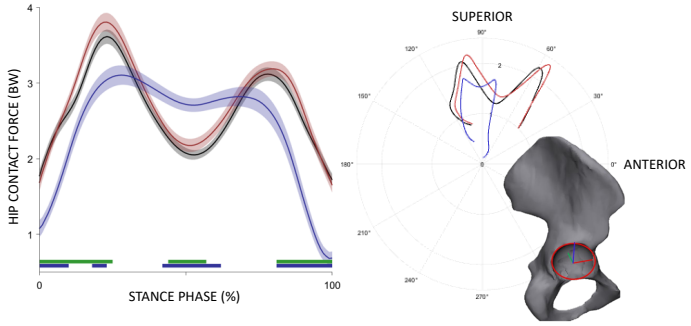

Figure 1: Ensemble average hip contact force magnitude (±1 standard error, left), and position and path (sagittal plane, right) for hip OA (blue), FAIS (red), and control (grey) groups during stance. Horizontal bars: periods of significant difference (P<.05) between hip OA/FAIS (green), hip OA/control (blue) groups.

Conclusions

Hip articular loading was not progressively lower or more concentrated from healthy controls to at-risk to established hip OA. Only those with hip OA had lower magnitude loading than healthy controls, which was located closer to the acetabular centre and constrained to a smaller region of cartilage. These hypothesis-generating findings suggest the biomechanical mechanisms of OA onset and progression could differ. Future investigation of tissue-level cartilage biomechanics (e.g., strains) may provide further insights into biomechanical markers across the disease spectrum.

References

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