INTRODUCTION

Knee osteoarthritis (KOA) is one of the most common joint diseases in the US, and is characterized by a progressive degeneration of the articular cartilage [1]. While the direct cause of KOA progression is unclear, obesity and female sex are significant risk factors for KOA development [2, 3]. Higher body mass index (BMI) and body fat mass are associated with abnormal cartilage composition and higher compressive forces at the knee joint [1, 4, 5]. Additionally, sex specific anthropometrics, may contribute to altered gait mechanics and knee joint loading in females. Furthermore, alterations in gait mechanics are implicated in KOA incidence and progression [1]. Specifically, individuals with KOA exhibit larger peak external knee adduction moments (KAM) and smaller external knee flexion moments (KFM), which contribute to disease progression [5, 6]. As such, abnormal sagittal and frontal knee joint loading in conjunction with obesity and female sex may predispose individuals to early onset of KOA. However, few data are available on the interaction of sex and obesity in the younger population despite their independent contributions to KOA risk. Therefore, the purpose of this study was to examine the influence of sex and BMI on knee joint sagittal and frontal plane gait mechanics. Dependent variables included knee flexion moment (KFM) and the first knee adduction moment peak (KAM1).

METHODS

Gait biomechanics were assessed in 42 obese (BMI≥30.0) and 39 normal weight (BMI 18.0-25.0) participants that were cohort matched on age and sex. Marker position and force plate data were sampled using a 9-camera motion capture system (Qualysis, Göteborg, Sweden) and 2 force plates (AMTI, Germantown MD). Participants completed 5 over-ground walking trials in laboratory standard neutral cushion footwear (Nike Pegasus, Beaverton, OR) at a self-selected speed. Speed was maintained within ±5% and monitored using infrared timing gates (Tractronix, Belton, MO) placed 2m apart surrounding the force plates. Marker position and force plate data were exported to Visual 3D, and low pass filtered using a 4th order zero-phase lag Butterworth at 6Hz and 50Hz, respectively. Stance phase was identified as the time-point between when the vertical GRF exceeded 20N and fell below 20N. A custom LabVIEW (National Instruments, Austin, TX) program was used to extract the peak external KFM during the first 50% of stance phase, and the first peak KAM. Joint moments were normalized to a product of bodyweight (N) and height (m) for analysis.

A 2 (BMI: normal, obese) by 2 (Sex: male, female) analysis of co-variance ($\alpha=0.05$) was used to examine dependent variables (KFM, KAM$_1$). Preliminary analyses indicated that self-selected gait speed was slower in the obese compared to normal weight group, and therefore, was used as a co-variate.

RESULTS AND DISCUSSION

The BMI by sex interaction was not significant for KAM1 ($F_{1,76}=.146, p=.703$) or KFM ($df=1, F=.329, p=.073$). A main effect was observed for sex in KAM1 ($F=3.879, p=.05$) and KFM ($F=8.776, p=.004$). After co-variying for gait speed, females exhibited larger KAM1 (.025 %BW•HT (95%CI: .022, .028) vs. .021 %BW•HT, (95%CI: .017, .024)
and lower KFM (0.052 %BW•HT, (95%CI: .047, .057) vs. .063 %BW•HT, (95%CI: .058, .069) compared to males.

There was no effect of BMI on KFM or KAM1 after co-varying for gait speed. This is contrary to previous findings in which obese individuals exhibited reduced peak knee extensor torque and larger KAM compared to normal weight participants at self-selected speed [4, 7]. Additionally, the discrepancy in findings may be explained by the difference in samples. Our obese sample is younger and with no previous clinical diagnosis of KOA. However, our results agree with a previous study in which no differences were found between younger obese individuals and normal controls [8].

Additionally, there was a significant main effect of sex on KFM and KAM1. Female sex is a significant risk factor for KOA incidence and aberrant biomechanics may be a key contributor. Females exhibited larger peak KAM1 compared to males, which contributes to preferential loading across the medial knee compartment. Previous studies have attributed lesser KAM in females to a more valgus knee alignment [9]. Additionally, greater KAM is attributed to greater varus alignment. However, we did not control or measure knee joint alignment, which may explain the discrepancies in our findings. Furthermore, females exhibited lower external KFM compared to males, which may indicate a quadriceps avoidance gait strategy [10] that is common among individuals with KOA. A quadriceps avoidance gait strategy may be explained by decreased quadriceps strength and muscle activity [10], which leads to decreased knee joint loading in the sagittal plane. A moderate amount of mechanical loading applied to the articular cartilage is necessary to maintain joint health, particularly in a normal joint environment absent of injury. [1]. Conversely, overloading or under-loading the cartilage may lead to degeneration of cartilage and development of OA [1]. As such, females may be at higher risk of OA due to lower sagittal and higher frontal plane knee joint loading regardless of the presence of obesity [3].

CONCLUSIONS

We observed no interaction between sex and BMI on KFM or KAM1. Furthermore, no differences were found in KFM or KAM1 between obese and normal weight participants. However regardless of BMI, females exhibited aberrant gait mechanics that are indicative of OA progression. Future studies should investigate the relationship between altered gait in females and knee cartilage morphology.

REFERENCES


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