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Gait biomechanics and hip muscular strength in patients with patellofemoral osteoarthritis

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ABSTRACT

A significant number of patients with patellofemoral osteoarthritis (PFOA) have described a history of patellofemoral pain syndrome (PFPS). This leads to speculation that the underpinning mechanical causes of PFPS and PFOA may be similar. Although alterations in gait biomechanics and hip strength have been reported in PFPS, this relationship has not yet been explored in PFOA. Therefore the purpose of this study was compare gait biomechanics and hip muscular strength between PFOA patients and a healthy control group. Fifteen patients with symptomatic, radiographic PFOA and 15 controls participated. All patients underwent a walking gait analysis and maximal hip strength testing. Biomechanical variables of interest included the peak angular values of contra-lateral pelvic drop, hip adduction and hip internal rotation during the stance phase. Hip abduction and external rotation strength were assessed using maximal voluntary isometric contractions. The PFOA group demonstrated significantly lower hip abduction strength compared to controls but no difference in hip external rotation strength. There were no statistical differences between the PFOA and control groups for contra-lateral pelvic drop, hip adduction and hip internal rotation angles during walking. Despite patients with PFOA exhibiting weaker hip abductor muscle strength compared to their healthy counterparts they did not demonstrate alterations in pelvis or hip biomechanics during gait. These preliminary data suggests that weaker hip abductor strength does not result in biomechanical alterations during gait in this population.

1. Introduction

Osteoarthritis (OA) is the most common joint disease in the world [1]. However, the aetiology of this disease remains unclear and there are currently no known treatments that have been proven to slow its progression. The knee is one of the most commonly affected joints and represents a major cause of pain and disability [2]. Traditionally, knee OA has been viewed as a disorder of the tibiofemoral joint, particularly of the medial compartment. However, studies have shown that 22–33% of knee OA patients exhibit osteoarthritic changes in the patellofemoral joint [3–5]. Additionally, compared with medial compartment OA, PFOA patients are more likely to report disability [4,5] and suffer an earlier onset of chronic symptoms [4,6].

Due to a current lack of literature investigating the biomechanical gait patterns associated with PFOA, it is pertinent to examine other patellofemoral disorders to help elucidate potential mechanisms. A study of PFOA patients waiting to undergo an arthroplasty showed that 22% of them described preceding patellofemoral pain syndrome (PFPS) in their adolescence and early adult years [6]. This finding is perhaps not surprising since up to 78% of PFPS patients still report chronic pain 5–20 years after rehabilitation [7–9]. The longevity of PFPS along with the low success rate following rehabilitation, leads to the hypothesis that the underpinning mechanical causes of PFPS and PFOA may be similar. This hypothesis is based on the premise that abnormal biomechanical patterns associated with the aetiology of PFPS may also contribute to degenerative changes at the patellofemoral joint over time.

Although the exact aetiology of PFPS remains unknown, some studies have shown excessive hip adduction and internal rotation during gait to be present in PFPS patients [10–12]. It is possible that abnormal hip mechanics are responsible for symptoms since several cadaveric studies have provided evidence for a link between abnormal lower extremity alignment and altered loading at the patellofemoral joint [13,14]. Excessive hip adduction may result in a medial collapse of the supporting limb and a theoretical increase in the quadriceps angle during stance (knee abduction).

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In turn, an increased quadriceps angle has been shown to result in elevated patellofemoral contact pressure [14]. Similarly, greater internal rotation of the femur was reported to also lead to increased patellofemoral contact pressure [13]. Therefore, alterations in gait kinematics could theoretically alter the contact pressure experienced in the patellofemoral joint, thus placing the underlying cartilage at risk for subsequent damage and degeneration.

Hip muscular strength has also received attention in the literature with respect to its association with PFPS. In particular, PFPS patients demonstrated decrements of 15–21% and 15–36% in hip abductor and hip external rotator strength respectively when compared to a healthy control group [15–17]. Moreover, reduced hip abductor and external rotator muscle strength has been associated with excessive hip abduction [16] and internal rotation [12] during gait respectively, suggesting that reduced hip muscle force output may be partly responsible for the atypical hip biomechanics.

Considering the epidemiological link between PFPS and PFOA, it is possible that the abnormal hip biomechanics and weakness of the hip musculature found in PFPS patients may also be contributing factors to PFOA. However, there is a dearth of literature examining muscular strength and gait biomechanics in patients with knee osteoarthritis whose symptoms originate primarily in the patellofemoral joint. Therefore, the purpose of this study was to investigate differences in hip strength and gait biomechanics between patients with mild to moderate PFOA and asymptomatic controls. It was hypothesised that compared to controls, PFOA patients would demonstrate greater hip adduction, hip internal rotation and contralateral pelvic drop during walking together with reduced hip abduction and external rotation muscular strength.

2. Methods

2.1. Subjects

Fifteen male and female subjects diagnosed with PFOA were recruited for the study. Fifteen gender matched asymptomatic subjects served as a control group (CON). Demographic data for all subjects can be found in Table 1. There were no significant differences between the PFOA and CON group in terms of age, mass or BMI. The sample size was selected following an a priori power analysis on the variable with the largest standard deviation (SD) noted in previous literature, peak hip internal rotation [12]. Using a within-group SD of 5.4° and expected difference between groups of 6.4°, a minimum of 12 subjects in each group were required to adequately power the study ($\alpha = 0.05$, $\beta = 0.8$). Prior to participation, all subjects provided written informed consent that had been approved by the Institutional Review Board.

Table 1
Mean (SD) average subject demographics, knee injury and osteoarthritis outcome score, and the frequency of compartmental OA radiographic scores.

	PFOA	CON	*P*
Demographics			
Gender distribution (female:male)	12:3	12:3	–
Age (years)	55 (9)	51 (9)	0.32
Mass (kg)	75.6 (10.5)	69.9 (13.3)	0.19
BMI (kg/m^2)	26.4 (3.7)	25.0 (3.5)	0.30
KOOS			
Pain (/100)	61.6 (12.5)	–	–
Symptoms (/100)	60.7 (19.5)	–	–
ADL (/100)	75.9 (13.4)	–	–
Sports (/100)	49.5 (26.9)		–
QOL (/100)	37.5 (19.4)	–	–
OA grade (KL)	PF compartment	TF compartment	
Grade 1	5	6	
Grade 2	5	4	
Grade 3	3	3	
Grade 4	2	0	

The PFOA participants were recruited through the university sports medicine centre while attending a knee OA clinic conducted by a sports medicine physician. Potential candidates that volunteered to participate in the study were evaluated by a certified Athletic Therapist and had to meet the following inclusion criteria: aged ≥40 years; knee pain originating primarily from the peri- or retro-patellar region; patellofemoral pain that was aggravated by at least two activities including stair ambulation, squatting, prolonged sitting, rising from seating, kneeling or exercise; radiographic evidence of OA (Kellgren–Lawrence grade ≥1) in the patellofemoral joint [18,19]; ability to walk without a cane or assistive device; familiar and comfortable with treadmill walking. Participants with unilateral or bilateral symptoms were included in the study. Exclusion criteria for the PFOA group included: prior history of patella fracture or recurrent subluxation; bony abnormalities including bone fracture, osteochondritis dissecans, or bi-partite patella; concomitant OA of the tibiofemoral joint that was more severe (greater K–L grade) than the patellofemoral joint; known OA of other lower extremity weight bearing joints (including the spine); knee, hip or ankle arthroplasty, osteotomy; arthroscopic surgery or knee injections within the last 3 months; currently undergoing (or within the last 6 weeks) physiotherapy for knee pain; any physical or medical problems for which strength testing/exercise would be contraindicated. A total of 19 volunteers were screened resulting in 4 being excluded from the study. The reasons for exclusion were tibiofemoral OA that was more severe than the patellofemoral joint ($n = 2$), evidence of hip OA ($n = 1$) and history of patellar fracture ($n = 1$).

The same exclusion criteria that was applied to the PFOA group was also used for the control group. In addition, all asymptomatic control subjects were required to meet the following inclusion criteria: aged ≥40 years; have no known OA in any lower extremity joint (including the spine); been free of any lower extremity musculoskeletal pain for the previous 6 months; were familiar and comfortable with treadmill walking.

2.2. Biomechanical measures

Biomechanical data were collected using an eight camera Vicon MX3 (Vicon Motion Systems, Oxford, UK) motion analysis. Twenty-one anatomical and 27 tracking markers placed bilaterally on the skin of the pelvis, thigh, shank and shoe of the participant (Fig. 1). Following a standing calibration trial the anatomical markers were removed and subjects walked on a treadmill for 3 min at a speed of 1.1 m/s while wearing standard, neutral, laboratory shoes (Nike Air Pegasus, Nike, Portland, OR). Treadmill walking was conducted due to space and setup restrictions imposed by the laboratory. The walking speed was selected to be similar to mean average treadmill walking speeds of knee OA patients in previous studies [20,21].

Following the 3-min treadmill accommodation period, kinematic data for ten footfalls were collected. Raw marker trajectory data were filtered using a fourth order low-pass Butterworth filter with cut-off frequency of 8 Hz [22]. Three-dimensional hip, knee and ankle joint angles were calculated using cardan angles with the distal segment expressed relative the proximal segment. Pelvic angles were defined as the pelvis segment relative to the laboratory. Visual 3D software (C-motion Inc, Germantown, MD) was used to filter all the marker data and calculate joint angles. Good reliability of this kinematic gait model has been documented previously [23]. Joint angle kinematics were analysed for the