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Hip muscle weakness in patients with symptomatic femoroacetabular impingement

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SUMMARY

Objective: Femoroacetabular impingement (FAI) is a pathomechanical process, which may cause hip pain, disability and early development of hip osteoarthritis (OA) in young and active adults. Patients with FAI experience functional disability during dynamic weight-bearing activities, which could originate from weakness of the hip muscles. The objective of this study was to compare hip muscle strength between patients with symptomatic FAI and healthy controls. It was hypothesized that patients would present overall hip muscle weakness compared to controls.

Methods: A total of 22 FAI patients and 22 controls matched for gender, age, and body mass participated in the study. We evaluated isometric maximal voluntary contraction (MVC) strength of all hip muscle groups using hand-held and isokinetic dynamometry, and electromyographic (EMG) activity of the rectus femoris (RF) and tensor fasciae latae (TFL) muscles during active flexion of the hip.

Results: FAI patients had significantly lower MVC strength than controls for hip adduction (28%), flexion (26%), external rotation (18%) and abduction (11%). TFL EMG activity was significantly lower in FAI patients compared with controls ($P = 0.048$), while RF EMG activity did not differ significantly between the two groups ($P = 0.056$).

Conclusions: Patients with symptomatic FAI presented muscle weakness for all hip muscle groups, except for internal rotators and extensors. Based on EMG recordings, it was demonstrated that patients with symptomatic FAI have a reduced ability to activate TFL muscle during hip flexion. These findings provide orthopedic surgeons with objective information about the amount and specificity of hip muscle weakness in patients with FAI. Future research should investigate the relationship between hip muscle weakness, functional disability and overuse injury risks, as well as the effects of hip muscle strengthening on clinical outcomes in individuals with symptomatic FAI.

Introduction

Femoroacetabular impingement (FAI) is a pathomechanical process that may instigate hip pain, disability and early development of hip osteoarthritis (OA) in young and active adults[1–3]. FAI mainly develops in individuals presenting aberrant morphologies of the proximal femur (cam FAI)[4] and/or acetabulum (pincer FAI)[5]. The Copenhagen Osteoarthritis Study revealed an incidence of cam deformities in 17% of men and 4% of women in a cohort of 3,202 unselected individuals[6]. These data have been recently confirmed in asymptomatic young adults. Hack *et al.* found an incidence of cam deformities in 25% of men and 5% of women[7]. In addition, Reichenbach *et al.* observed an incidence of 24% in men[8]. Although the combination of cam and pincer deformities is the most frequent FAI finding[9,10], pure cam deformities seem to be more common in men, while pure pincer deformities are seen in women[9]. These specific femoral and/or acetabular deformities may cause abnormal mechanical contacts to occur between the hip joint structures, particularly at terminal hip flexion and internal rotation[4,11]. Consequent repetitive shear forces and impacts may degenerate the acetabular labrum and/or cartilage[11], thereby causing hip pain. Pain is primarily localized in the anteromedial groin[12], mainly induced by static and dynamic situations which involve hip flexion[12], and exacerbated by moderate-to-high-intensity physical tasks[1,2]. Therefore, subjects with FAI experience disability and limitations while performing activities of daily living, and these symptoms could even worsen during demanding physical activities[13,14].

In the last few years, objective measures of physical function have been increasingly implemented in patients with FAI[15–17], and research has shown FAI-related kinematic alterations of the

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symptomatic lower limb during dynamic weight-bearing activities. Kennedy *et al.* found that patients with FAI presented decreased frontal and sagittal hip range of motion and attenuated frontal pelvic mobility during level gait at a self-selected normal speed[15]. Austin *et al.* observed excessive hip adduction and internal rotation with related hip pain during moderate-to-high-intensity eccentric activities in a female patient with FAI[16]. Taken as a whole, these kinematic alterations could result from hip muscle weakness[15,18]. Nevertheless, to our knowledge hip muscle strength has not yet been objectively investigated in this patient population. In fact, no information is actually available about specific hip muscle weakness expectations in patients with FAI, despite the fact that hip muscle strength evaluation has been recommended to complete physical examination in patients with a painful hip[19]. For these reasons, it is of primary importance that orthopedic surgeons, rheumatologists and physical therapists are aware of the amount and specificity of hip muscle weakness – and eventually of functional disabilities and injury risks secondary to hip muscle weakness – in patients with FAI.

Therefore, the objective of this study was to compare hip muscle strength and hip flexor electromyographic (EMG) activity during active flexion of the hip between patients with symptomatic FAI and age-matched healthy controls. It was hypothesized that patients with FAI would present significant overall hip muscle weakness and reduced hip flexor muscle activation compared to controls.

Methods

Subjects

A total of 22 patients (14 women) with FAI aged between 20 years and 50 years, and 22 healthy controls matched for gender, age, and body mass volunteered to participate in the study. Age [FAI: 32 ± 9 years; controls: 32 ± 9 years; mean $\pm$ standard deviation (SD)], height (FAI: 175 ± 8 cm; controls: 171 ± 6 cm) and body mass (FAI: 72 ± 14 kg; controls: 69 ± 11 kg) were comparable between the two groups. All patients, who were scheduled for surgery at the point of recruitment, had a FAI diagnosis carried out by the same senior surgeon (ML) using clinical, radiographic and magnetic resonance imaging assessments. Patients presented different types of FAI: six had cam, four had pincer, and 12 had combined FAI. Although eight patients had bilateral FAI diagnosis, only the most symptomatic side was considered. More than two-third of patients with FAI were involved in recreational physical activities (three sessions/week, minimum 30 min) before the onset of hip pain. Control subjects were asymptomatic and had no history of hip pain. Their average physical activity level was comparable to that of FAI patients. For both groups, subjects presenting any disorder to the lower extremities (excepting hip impingement in the FAI group) that would have negatively influenced muscle strength evaluation were not included in the study.

Since no hip muscle strength data of individuals with FAI were found in the literature, sample size was determined on the basis of hip flexor muscle weakness detected in patients with hip OA compared with matched controls[20]. Power analysis indicated that a sample size of 18 subjects/group was required to detect significant hip flexor strength differences between FAI and controls (effect size $= 0.86$; $\alpha = 0.05$; power $= 0.8$). The study was conducted according to the Helsinki declaration of 1975, as revised in 2000, and the protocol was approved by the Ethics Committee of the Canton of Zurich (Switzerland). All the subjects signed a written informed consent before participating in the study.

Experimental procedures

Isometric maximal voluntary contraction (MVC) strength of the hip abductor, adductor, internal rotator, external rotator, flexor and extensor muscles was measured unilaterally during a 90-min testing session. Muscle weakness was quantified by comparing strength outcomes between patients with FAI and controls (between-subject comparison)[21]. For patients, the symptomatic (unilateral FAI) or the most symptomatic (bilateral FAI) hip was tested, while controls had the respective hip evaluated, according to lower limb dominance (kicking limb). For practical reasons, hip abduction, adduction, internal rotation and external rotation were randomly tested first (restricted randomization, random number table). Subsequently, hip flexion and extension were assessed in a randomized order. In a subgroup of patients ($N = 16$, nine women) and in their corresponding controls, muscle activation of the rectus femoris (RF) and tensor fasciae latae (TFL) muscles (i.e., the two main superficial hip flexor muscles), was measured by means of surface EMG during active flexion of the hip. A single investigator (NCC), who was not blind to participants' characteristics, conducted all the assessments with the assistance of two other co-investigators (JFIG, SS).

Hip muscle strength

Hip abduction, adduction, internal rotation and external rotation MVC strength were evaluated with hand-held dynamometry (Nicholas Manual Muscle Tester, Lafayette Inc., Lafayette, IN, USA), which showed good-to-excellent intra-rater reliability for the test positions used in this study[22,23]. For hip abduction and adduction, subjects laid on their side on a treatment table; the tested hip was at 0° of hip flexion, extension and rotation, with the ipsilateral knee fully extended. For hip abduction, subjects laid on the non-tested side; the tested hip was abducted to approximately 10°, and the contralateral hip and knee were flexed to 45° and 60°, respectively, to provide comfort and stabilization[24]. Prior to hip abduction testing, the mass of the tested limb was measured by positioning the dynamometer pad 5 cm proximal to the medial malleolus, so as to correct for gravity. For hip adduction, subjects were lying on the tested side; the tested limb laid on the treatment table, and the contralateral limb rested on a padded box with hip and knee flexed to 45° and 60°, respectively[22]. For hip internal and external rotation, subjects sat on the treatment table, with the two legs hanging free over the edge[23,25], and hips and knees flexed at 90°. The dynamometer pad was held by the investigator 5 cm proximal to the lateral (abduction and internal rotation) and medial (adduction and external rotation) malleolus[22,23,25].

Hip flexion and extension MVC strength was assessed using isokinetic dynamometry (Biodex System 2, Biodex Medical Systems, New York, USA) to simultaneously record MVC torque and EMG traces (Fig. 1). Subjects laid supine on the dynamometer chair with the chair back inclined to 15° and the dynamometer rotation axis aligned to the hip rotation center (greater trochanter)[26] (Fig. 2). The tested hip was flexed to 45° and the ipsilateral thigh was strapped to the dynamometer pad, approximately 5 cm proximal to the lateral femoral condyle. The pelvis and trunk were secured to the dynamometer chair with straps. Prior to testing, the mass of the tested limb was measured to correct for gravity.

For each muscle group, subjects completed two submaximal familiarization trials followed by 3–4 MVC trials (not more than 10% of difference between the two highest MVC was tolerated), during which they were asked to perform maximal efforts for 3–4 s, without any concern to rate of force development. The rest interval between trials was 60 s. Standardized verbal encouragement was consistently provided by the investigators. The main strength outcome was MVC

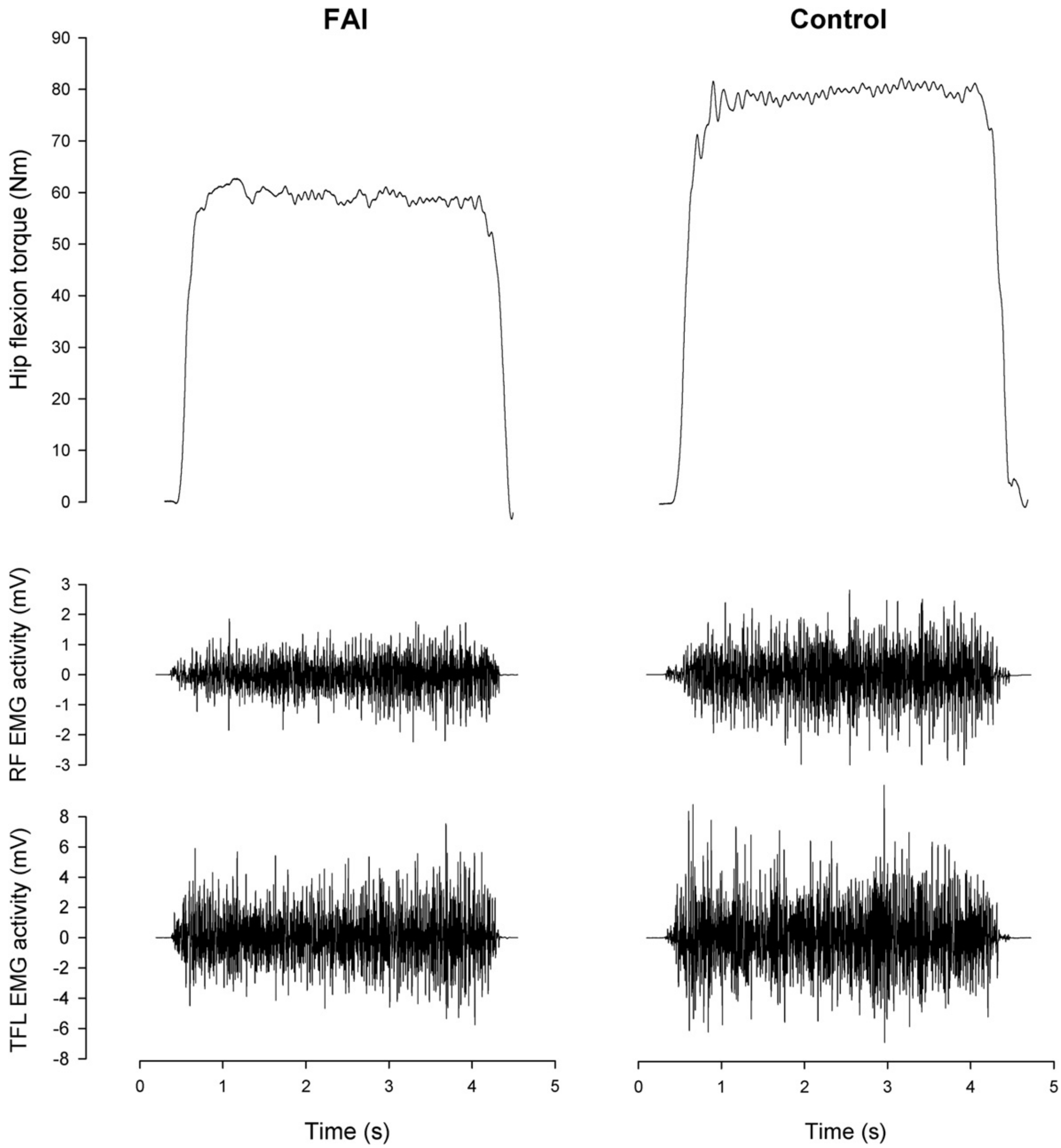

Fig. 1. Experimental traces of hip flexion torque and EMG activity in one representative patient with FAI (left panels) and in his respective control (right panels).

torque normalized to body mass[27]. Since the hand-held dynamometer measured force in Newtons, force was multiplied by the lever arm length to obtain torque. For hip abduction and adduction, the lever arm length corresponded to the distance between the greater trochanter and 5 cm proximal to the lateral malleolus. For hip internal and external rotation, lever arm length corresponded to the distance between the lateral femoral condyle and 5 cm proximal to the lateral malleolus. For each muscle group, only the highest MVC was retained for analyses. To better characterize muscle weakness, percentage differences in MVC strength between FAI and controls were calculated as (100 × (MVC strength of FAI−MVC strength of controls)/MVC strength of controls).

After each MVC trial, both patients and controls were asked to quantify hip joint pain by placing a vertical mark on a 100-mm horizontal line, known as the visual analog scale (VAS)[28]. The line ranged from 0 (no pain at all) to 100 (not endurable pain). For each muscle group, the mean pain score was retained for analysis.

Hip flexor EMG activity

Two pairs of silver-chloride surface electrodes (inter-electrode distance of 25 mm) were positioned on the RF and TFL muscles according to standard recommendations[29]. For RF, electrodes were placed at 50% on the line from the anterior superior iliac spine to the upper border of the patella. For TFL, electrodes were placed proximally at 17% on the line from the anterior superior iliac spine to the lateral femoral condyle. Low resistance between the two electrodes was achieved with light abrasion of the skin and cleaning with alcohol. The ground electrode was positioned on the ipsilateral patella. EMG signals were amplified with a bandwidth frequency ranging from 10 Hz to 500 Hz (gain 1000), digitized online at a sampling frequency of 2 kHz, and recorded by the Biopac system (MP150, Biopac System Inc., Goleta, CA, USA). For both RF and TFL muscles, EMG root mean square amplitude was calculated during 500 ms around hip flexion MVC torque[30], using a window

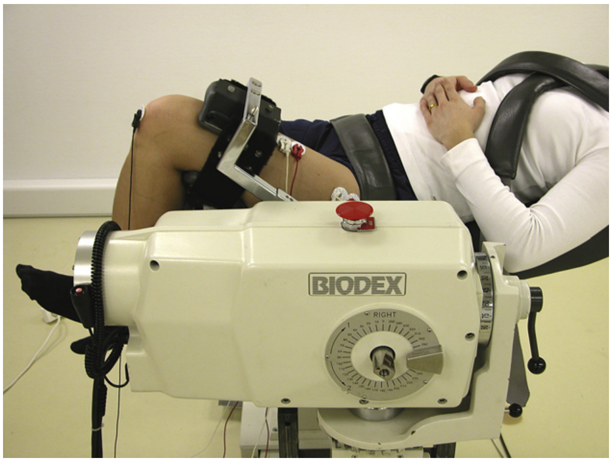

Fig. 2. Experimental test set-up for hip flexor and extensor isometric strength assessments. The tested hip was flexed at 45° with the thigh strapped to the dynamometer pad. Surface EMG electrodes were positioned on the RF and TFL muscles.

length of 125 ms. Only the MVC trial associated to the highest EMG root mean square amplitude was retained for analyses.

Statistical analysis

Normal distribution of the data was assessed using Shapiro–Wilk tests. Between-group (FAI vs controls) differences in MVC strength for each of the hip muscle groups, and hip flexor EMG activity were investigated with paired *t*-tests in case of normal distribution[31]. In case of non-normal distribution, Wilcoxon signed-rank tests were used. Statistical analyses were performed with SPSS 18.0 software (SPSS Inc., Chicago, IL, USA). The significance level was set at $P < 0.05$.

Results

Patients with FAI had significantly lower MVC strength than controls for hip adduction (28%, $P = 0.003$), flexion (26%, $P = 0.004$; see also Fig. 1), external rotation (18%, $P = 0.04$) and abduction (11%, $P = 0.03$), while hip internal rotation and extension did not differ significantly between the two groups (Table I). Hip joint VAS pain scores reported by patients during MVC trials ranged between 18 mm and 27 mm (Table I), while controls reported no hip joint pain.

EMG activity of the TFL muscle was significantly lower in patients than in controls ($P = 0.048$; Table II), while RF activity did not differ significantly between the two groups ($P = 0.056$).

Discussion

Patients with FAI showed significant muscle weakness compared to asymptomatic controls for hip flexion, hip adduction, hip external rotation and hip abduction. Hip flexor muscle weakness was accompanied by reduced muscle activation in patients, as witnessed by the lower EMG activity of TFL in comparison to controls.

The mean hip muscle weakness observed in our patients with FAI (16%, average of all hip muscle groups) was slightly smaller than the weakness reported by Rasch *et al.*[32] (20%) and by Arokoski *et al.*[20] (23%) in patients with hip OA, when compared to healthy controls. If one supposes that muscle weakness is a marker of OA progression[33], these results agree with the assumption that FAI could be a process potentially leading to hip OA[1–3]. Considering single hip muscle groups, the individuals with FAI tested in this study had less hip abductor and extensor weakness, but similar hip flexor weakness compared to patients with hip OA tested in previous studies[20,32]. Regarding hip adductor muscle strength in patients with hip OA, contradictory results have been reported in the literature. Similarly to the current FAI patients, Arokoski *et al.* found that patients with hip OA had 25% hip adductor weakness compared to healthy controls[20]. In contrast, Rasch *et al.* found no hip adductor strength difference between hip OA patients and healthy controls[32]. Based on the current results, hip flexor and adductor muscles of patients with FAI showed the greatest levels of muscle weakness, well beyond the 20% threshold[27]. It is tempting to suggest that an assessment of hip flexor and adductor muscle strength could be included in routine clinical examinations besides the ordinary evaluations (e.g., impingement test, hip range of motion, investigation of symptoms) to help diagnose FAI[19]. This could be easily performed using the procedures described in this study, entailing the utilization of a hand-held dynamometer, which is the most practical tool for measuring hip muscle strength in clinical practice[27]. In case of unilateral FAI, muscle weakness can be calculated as the strength difference between the involved and uninvolved hip (within-subject comparison). However, since FAI frequently presents bilaterally[12], we recommend the assessment of muscle strength on the most symptomatic hip, and the comparison with a healthy control group (between-subject comparison). Using this approach, hip muscle weakness greater than 20% could be arbitrarily viewed as “almost certainly pathological”[27].

The considerable FAI-related hip muscle weakness observed in this study could potentially originate from different factors: a mechanical/anatomical limit[34], reduced muscle mass (atrophy) or reduced muscle activation (possibly related to pain and/or fear of pain[20]) during MVC compared to controls[35]. We can *a priori* exclude that a mechanical/anatomical limit within the hip joint impaired the ability of patients to develop strength, since all assessments were completed in isometric conditions at joint angles far away

Table I
Hip muscle strength and hip joint pain during hip muscle strength assessment in patients with FAI ($N = 22$) and healthy controls ($N = 22$)

	Normalized torque (Nm/kg)		*P* value	Difference (%)	VAS (0–100 mm)
	Mean ± SD			Mean ± 95% CI	Mean ± SD
	FAI	Controls		FAI vs. Controls	FAI
Hip adduction	1.57 ± 0.82	2.17 ± 0.49	0.003	28 ± 17	22 ± 24
Hip abduction	1.81 ± 0.43	2.03 ± 0.31	0.028	11 ± 11	21 ± 25
Hip internal rotation	0.47 ± 0.16	0.55 ± 0.17	0.076	14 ± 19	25 ± 22
Hip external rotation	0.46 ± 0.21	0.56 ± 0.15	0.040	18 ± 21	23 ± 23
Hip flexion	0.87 ± 0.46	1.17 ± 0.37	0.004	26 ± 18	27 ± 26
Hip extension	1.64 ± 1.00	1.66 ± 0.86	0.592	1 ± 30	18 ± 20

CI: confidence interval, 0 = no pain at all, 100 = not endurable pain. Controls reported no hip joint pain.

Table II
Hip flexor EMG activity in patients with FAI ($N = 16$) and healthy controls ($N = 16$) during active flexion of the hip

	EMG root mean square (μV)		*P* value
	Mean ± SD		
	FAI	Controls	
RF	186 ± 131	294 ± 184	0.056
TFL	401 ± 251	582 ± 323	0.048

from terminal range of motion (e.g., hip flexion at 45°, hip adduction at 0°), where FAI might cause abnormal bony contacts between the femoral head–neck junction and the acetabulum rim to occur[34]. Hip flexor (psoas and RF muscle) and adductor muscle mass has been found to be reduced in the involved side in comparison to the uninvolved side in patients with unilateral hip OA[36]. Due to the above-discussed similarities between patients with FAI and OA, muscle atrophy cannot therefore be excluded as a potential contributor to muscle weakness in the current patient group, even though no attempt was made to quantify hip muscle mass in the present study. On the other hand, the lower EMG activity of the TFL muscle observed in patients compared to controls revealed an impaired ability to voluntarily activate this hip flexor muscle in people with FAI, which could contribute, at least in part, to muscle weakness.

Recent research has focused on lower limb kinematics of patients with FAI during dynamic weight-bearing activities[15,16]. Kennedy *et al.* compared hip and pelvic kinematics of FAI patients to those of healthy controls during level gait at a self-selected normal speed[15]. They showed that patients presented reduced peak hip abduction angle and less total frontal hip range of motion in the symptomatic hip in comparison to controls. As argued by Kennedy *et al.*[15] and consistent with the results of the present study, these alterations could be the result of a strategy adopted by patients to compensate for a hip muscle function deficiency. In a similar way, growing evidence suggests that hip muscle weakness may alter lower limb kinematics in patients with patellofemoral pain syndrome[18]. During weight-bearing activities, the external force moments acting on the lower extremities induce femoral adduction, internal rotation and flexion. This triplanar motion is most commonly observed during the weight acceptance phase of high-demanding eccentric activities, such as running or landing from a jump. If hip muscles – in particular hip abductors and external rotators[37] – are not strong enough to counteract the external force moments, excessive femoral adduction and internal rotation would lead to an increased dynamic valgus of the knee and decreased patellofemoral joint contact area, which are suggested as possible factors leading to patellofemoral overuse injury and pain. In a single case study, excessive hip adduction and internal rotation (i.e., dynamic valgus of the knee) have also been observed in a 25-years-old female patient with symptomatic FAI during a single-limb step down, running, and drop jump[16]. These hip motion alterations also reproduced hip joint pain in that patient. Thus, FAI-related hip muscle weakness could result in lower limb kinematic alterations during moderate-to-high intensity dynamic weight-bearing activities, which could cause functional disability. These alterations seem to exacerbate the symptoms probably due to the increased anteromedial contact stress in the femoroacetabular joint[38], where bony contact and joint damage occurs. Additionally, it is not excluded that these kinematic alterations could lead to patellofemoral pain in patients with FAI. This would explain why in a recent study of Clohisy *et al.* 27% (14/51) of patients with FAI reported knee pain at clinical examination[39]. Future research is however needed to objectively investigate the relationships between hip muscle weakness, dynamic malalignement of the lower extremities during weight-bearing activities, functional disability and overuse injuries in patients with FAI.

The current study presents some potential limitations. No distinction was made between the different types of FAI (cam, pincer, combined). However, *a posteriori* statistical comparison showed no significant differences in hip muscle weakness between the three subgroups of patients. Hand-held instead of stabilized dynamometry was used for strength assessment of hip abduction, adduction, internal rotation and external rotation. Intra-rater reliability of hand-held dynamometry has been shown to be lower compared to stabilized dynamometry due to the influence of the investigator's strength to resist the measured forces[40]. Nevertheless, hand-held dynamometry assessments are more clinically feasible, showed good-to-excellent reliability for the hip movements that we tested[22,23] and the investigator had no difficulty to resist the maximal hip muscle strength of patients. Due to time constraints, we only recorded EMG activity of two superficial hip flexor muscles. Therefore, these EMG results cannot be generalized to all hip muscle groups.

In conclusion, patients with symptomatic FAI presented significant muscle weakness for all hip muscle groups, except internal rotators and extensors. Based on EMG recordings, it was demonstrated that patients with FAI have a reduced ability to activate TFL muscle. These findings have important implications for orthopedic surgeons and rheumatologists as they provide objective information about the amount and specificity of hip muscle weakness in patients with FAI, which could be helpful for early recognition of the pathology. Long-term benefits of conservative treatment (e.g., physical therapy) in patients with FAI are usually questionable[41]. On the contrary, some preliminary results show that in the short-term the improvement of hip control – by way of external assistance devices[16] or strength and coordination training[38] – could alleviate the symptoms and improve the clinical outcomes. Therefore, the effects of a preoperative hip strengthening program on the clinical outcomes of patients with FAI would be worthwhile to investigate.

Author contributions

Conception and design: NCC, NAM, MB, ML. Analysis and interpretation of the data: NCC, NAM, FMI. Drafting of the article: NCC, NAM, MB, ML. Critical revision of the article for important intellectual content: NCC, NAM, JFIG, SS, MB, FMI, ML. Final approval of the article: NCC, NAM, JFIG, SS, MB, FMI, ML. Provision of study materials or patients: NCC, ML. Statistical expertise: NCC, NAM, FMI. Administrative, technical, or logistic support: NCC. Collection and assembly of data: NCC, JFIG, SS.

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Conflict of interest

The authors have no conflict of interest.

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References

1. Ganz R, Parvizi J, Beck M, Leunig M, Nötzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res 2003;417:112–20.

2. Leunig M, Beck M, Dora C, Ganz R. Femoroacetabular impingement: etiology and surgical concept. Oper Tech Orthop 2005;15:247–55.
3. Leunig M, Beaulé PE, Ganz R. The concept of femoroacetabular impingement: current status and future perspectives. Clin Orthop Relat Res 2009;467:616–22.
4. Ito K, Minka- II MA, Leunig M, Werlen S, Ganz R. Femoroacetabular impingement and the cam-effect. A MRI-based quantitative anatomical study of the femoral head-neck offset. J Bone Joint Surg Br 2001;83:171–6.
5. Siebenrock KA, Schoeniger R, Ganz R. Anterior femoroacetabular impingement due to acetabular retroversion: treatment with periacetabular osteotomy. J Bone Joint Surg Am 2003;85:278–86.
6. Gosvig KK, Jacobsen S, Sonne-Holm S, Gebuhr P. The prevalence of cam-type deformity of the hip joint: a survey of 4151 subjects of the Copenhagen Osteoarthritis Study. Acta Radiol 2008;49:436–41.
7. Hack K, Di Primio G, Rakhra K, Beaulé PE. Prevalence of cam-type femoroacetabular impingement morphology in asymptomatic volunteers. J Bone Joint Surg Am 2010;92:2436–44.
8. Reichenbach S, Jüni P, Werlen S, Nüesch E, Pfirrmann CW, Trelle S, *et al*. Prevalence of cam-type deformity on hip magnetic resonance imaging in young males: a cross-sectional study. Arthritis Care Res (Hoboken) 2010;62:1319–27.
9. Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br 2005;87:1012–8.
10. Allen D, Beaulé PE, Ramadan O, Doucette S. Prevalence of associated deformities and hip pain in patients with cam-type femoroacetabular impingement. J Bone Joint Surg Br 2009;91:589–94.
11. Tannast M, Goricki D, Beck M, Murphy SB, Siebenrock KA. Hip damage occurs at the zone of femoroacetabular impingement. Clin Orthop Relat Res 2008;466:273–80.
12. Beaulé PE, Allen DJ, Clohisy JC, Schoenecker P, Leunig M. The young adult with hip impingement: deciding on the optimal intervention. J Bone Joint Surg Am 2009;91:210–21.
13. Philippon MJ, Maxwell RB, Johnston TL, Schenker M, Briggs KK. Clinical presentation of femoroacetabular impingement. Knee Surg Sports Traumatol Arthrosc 2007;15:1041–7.
14. Bizzini M, Nötzli HP, Maffiuletti NA. Femoroacetabular impingement in professional ice hockey players: a case series of 5 athletes after open surgical decompression of the hip. Am J Sports Med 2007;35:1955–9.
15. Kennedy MJ, Lamontagne M, Beaulé PE. Femoroacetabular impingement alters hip and pelvic biomechanics during gait: walking biomechanics of FAI. Gait Posture 2009;30:41–4.
16. Austin AB, Souza RB, Meyer JL, Powers CM. Identification of abnormal hip motion associated with acetabular labral pathology. J Orthop Sports Phys Ther 2008;38:558–65.
17. Lamontagne M, Kennedy MJ, Beaulé PE. The effect of cam FAI on hip and pelvic motion during maximum squat. Clin Orthop Relat Res 2009;467:645–50.
18. Powers CM. The influence of abnormal hip mechanics on knee injury: a biomechanical perspective. J Orthop Sports Phys Ther 2010;40:42–51.
19. Kelly BT, Williams III RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med 2003;31:1020–37.
20. Arokoski MH, Arokoski JP, Haara M, Kankaanpää M, Vesterinen M, Niemitukia LH, *et al.* Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. J Rheumatol 2002;29:2185–95.
21. Hinman RS, Hunt MA, Creaby MW, Wrigley TV, McManus FJ, Bennell KL. Hip muscle weakness in individuals with medial knee osteoarthritis. Arthritis Care Res (Hoboken) 2010;62:1190–3.
22. Krause DA, Schlagel SJ, Stember BM, Zoetewey JE, Hollman JH. Influence of lever arm and stabilization on measures of hip abduction and adduction torque obtained by hand-held dynamometry. Arch Phys Med Rehabil 2007;88:37–42.
23. Pua YH, Wrigley TV, Cowan SM, Bennell KL. Intrarater test-retest reliability of hip range of motion and hip muscle strength measurements in persons with hip osteoarthritis. Arch Phys Med Rehabil 2008;89:1146–54.
24. Widler KS, Glatthorn JF, Bizzini M, Impellizzeri FM, Munzinger U, Leunig M, *et al*. Assessment of hip abductor muscle strength: a validity and reliability study. J Bone Joint Surg Am 2009;91:2666–72.
25. Magalhães E, Fukuda TY, Sacramento SN, Forgas A, Cohen M, Abdalla RJ. A comparison of hip strength between sedentary females with and without patellofemoral pain syndrome. J Orthop Sports Phys Ther 2010;40:641–7.
26. Frost KL, Bertocci GE, Wassinger CA, Munin MC, Burdett RG, Fitzgerald SG. Isometric performance following total hip arthroplasty and rehabilitation. J Rehabil Res Dev 2006;43:435–44.
27. Maffiuletti NA. Assessment of hip and knee muscle function in orthopaedic practice and research. J Bone Joint Surg Am 2010;92:220–9.
28. Huskisson EC. Measurement of pain. Lancet 1974;2:1127–31.
29. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. J Electromyogr Kinesiol 2000;10:361–74.
30. Missenard O, Mottet D, Perrey S. Factors responsible for force steadiness impairment with fatigue. Muscle Nerve 2009;40:1019–32.
31. Bland JM, Altman DG. Matching. BMJ 1994;309:1128.
32. Rasch A, Dalén N, Berg HE. Test methods to detect hip and knee muscle weakness and gait disturbance in patients with hip osteoarthritis. Arch Phys Med Rehabil 2005;86:2371–6.
33. Brandt KD. Is a strong quadriceps muscle bad for a patient with knee osteoarthritis? Ann Intern Med 2003;138:678–9.
34. Wyss TF, Clark JM, Weishaupt D, Nötzli HP. Correlation between internal rotation and bony anatomy in the hip. Clin Orthop Relat Res 2007;460:152–8.
35. Mizner RL, Petterson SC, Stevens JE, Vandenborne K, Snyder-Mackler L. Early quadriceps strength loss after total knee arthroplasty. The contributions of muscle atrophy and failure of voluntary muscle activation. J Bone Joint Surg Am 2005;87:1047–53.
36. Rasch A, Byström AH, Dalén N, Berg HE. Reduced muscle radiological density, cross-sectional area, and strength of major hip and knee muscles in 22 p.tients with hip osteoarthritis. Acta Orthop 2007;78:505–10.
37. Ireland ML, Willson JD, Ballantyne BT, Davis IM. Hip strength in females with and without patellofemoral pain. J Orthop Sports Phys Ther 2003;33:671–6.
38. Yazbek PM, Ovanessian V, Martin RL, Fukuda TY. Non-surgical treatment of acetabular labrum tears: a case series. J Orthop Sports Phys Ther 2011;10.2519/jospt.2011.3225
39. Clohisy JC, Knaus ER, Hunt DM, Lesher JM, Harris-Hayes M, Prather H. Clinical presentation of patients with symptomatic anterior hip impingement. Clin Orthop Relat Res 2009;467:638–44.
40. Scott DA, Bond EQ, Sisto SA, Nadler SF. The intra- and inter-rater reliability of hip muscle strength assessments using a handheld versus a portable dynamometer anchoring station. Arch Phys Med Rehabil 2004;85:598–603.
41. Parvizi J, Leunig M, Ganz R. Femoroacetabular impingement. J Am Acad Orthop Surg 2007;15:561–70.