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A Comparative Analysis of Lower Limb Muscle Activation and Co-Contraction Patterns During Gait in Individuals With Patellofemoral Pain Syndrome

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Abstract

Background. Patellofemoral pain syndrome (PFPS) is a prevalent musculoskeletal condition that disrupts functional activities like walking and stair climbing due to anterior knee pain. While muscle weakness has traditionally been emphasised, growing evidence suggests that impaired neuromuscular coordination may also play a critical role in PFPS-related gait dysfunction.

Aim. This study aimed to compare lower-limb muscle activation patterns and co-contraction behaviour during gait between individuals with PFPS and healthy controls, focusing on both timing and intensity of activation across key knee and ankle muscle groups.

Methods. Fifteen PFPS patients and fifteen age- and activity-matched healthy controls walked at a self-selected pace while surface electromyography (EMG) data were recorded from eight lower-extremity muscles. EMG signals were processed to extract normalised peak activation timing, magnitude, and co-contraction indices (CCI) for knee and ankle joints. Between-group comparisons were conducted using independent *t*-tests and Pearson correlation analyses.

Results. The PFPS group exhibited significantly earlier (VL: 13% vs. 100%; RF: 4% vs. 98% of stance, both $p < 0.05$) and greater peak activation (VL: 0.202 vs. 0.112; RF: 0.109 vs. 0.062, both $p < 0.05$) in the quadriceps. They also showed delayed hamstring (ST: 100% vs. 3% of stance, $p < 0.05$) activation and increased medial gastrocnemius amplitude (GM: 0.282 vs. 0.161, $p < 0.05$). Despite these timing shifts, average CCI values did not differ significantly between groups. However, flexor–extensor activation synchrony was notably higher in PFPS ($r \approx 0.82$) compared with controls ($r \approx 0.39$).

Conclusions. Individuals with PFPS demonstrate altered neuromuscular control during gait, including phase-shifted activation of knee muscles and modified ankle muscle recruitment. These changes may elevate joint stress and contribute to persistent pain. Rehabilitation programmes should emphasise not only muscle strengthening but also retraining of activation timing and coordination to promote more efficient and pain-free gait patterns.

Keywords: patellofemoral pain syndrome; gait; electromyography; muscle activation; co-contraction

1. INTRODUCTION

Patellofemoral pain syndrome (PFPS) is one of the most common musculoskeletal disorders of the knee, affecting up to ~22% of the general population annually (and nearly 30% of adolescents) (Xu et al., 2024). Lifetime prevalence estimates approach 25% (Glaviano et al., 2015), underscoring the substantial clinical impact of PFPS among young and active individuals. PFPS is characterised by diffuse anterior knee pain, often exacerbated by activities like squatting, stair ambulation, or running (Santilli et al., 2024). Despite its high frequency, the precise biomechanics and pathomechanics underlying PFPS remain incompletely understood (Choi et al., 2025). Multifactorial causes have been proposed, including quadriceps muscle weakness (particularly of the vastus medialis oblique), patellar maltracking, tight lateral retinaculum, abnormal foot biomechanics, and muscular imbalances around the knee (Collado & Fredericson, 2010). In particular, an imbalance in activation between the vastus medialis and vastus lateralis has long been thought to contribute to lateral patellar deviation and PFPS symptoms (Hosseini & Farahmand, 2024).

Given the importance of dynamic knee stability during functional activities, there is growing interest in the neuromuscular control of the lower limb in PFPS. Modern gait analysis techniques offer insights into subtle alterations in movement and muscle function that may not be apparent on static examination (Choi et al., 2025).

Prior electromyographic studies in PFPS have often focused on isolated aspects such as the timing of vasti muscle activation or the ratio of vastus medialis to lateralis activation (Kim & Song, 2012). For example, delayed onset of the vastus medialis relative to the vastus lateralis and other timing asymmetries have been reported in those with PFPS (Ferrari et al., 2014). Additionally, interventions like therapeutic taping that alter patellar alignment have been shown to modify the activation timing of the vasti muscles in PFPS patients (Bennell et al., 2006), reinforcing the idea that muscle activation patterns are linked to patellofemoral joint mechanics.

However, relatively few studies have comprehensively examined the full lower-extremity muscle activation patterns during gait in PFPS or evaluated how concurrent activation (co-contraction) of muscle groups may differ from pain-free individuals. Understanding these patterns is important because gait is a fundamental daily activity commonly limited by PFPS pain, and altered gait mechanics could contribute to abnormal patellofemoral joint loading.

In knee osteoarthritis and ligament injury populations, increased antagonist co-contraction is a known compensatory strategy to stabilise the joint (Ford et al., 2008). It is plausible that patients with PFPS might also adopt altered co-activation strategies (e.g. greater simultaneous quadriceps and hamstrings activity) to improve knee stability due to pain or perceived instability, but evidence is limited and mixed. Some modelling work suggested PFPS patients experience greater patellofemoral joint forces partly due to heightened quadriceps–hamstrings co-contraction during walking (Besier et al., 2009).

Overall, there is a need for a detailed analysis of lower-limb muscle activation and co-contraction patterns in PFPS during walking. While previous studies often focused on individual muscles or non-functional tasks, our study uniquely investigates coordinated activation and antagonist co-contraction across multiple muscle groups during overground gait. By examining both timing and amplitude of activation, as well as flexor–extensor synchrony, we aim to clarify neuromuscular alterations that may underlie joint stress in PFPS. Such knowledge has direct clinical relevance, as it can inform rehabilitation programmes that target not only muscle strength but also activation timing and coordination, ultimately helping clinicians design more effective gait retraining and pain reduction strategies for patients. Therefore, the purpose of this study was to compare lower-limb muscle activation profiles and co-contraction behaviour between individuals with PFPS and healthy controls. We hypothesised that the PFPS group

would show altered activation timing and amplitudes – especially in quadriceps and hamstrings – and increased antagonist co-contraction.

2. METHODS

Participants. The required sample size for this study was calculated using G*Power software (*version 3.1.9.2, Heinrich Heine University, Düsseldorf, Germany*). Estimation was informed by a previous study by Chang et al. (2015), which investigated electromyographic activity of the vastus medialis obliquus (VMO) and vastus lateralis (VL) muscles in PFPS patients during sling-based exercises. In their pilot analysis of seven PFPS patients, the mean differences in EMG variables showed standard deviations ranging from 0.09 to 0.16, corresponding to a medium effect size (Cohen's $d \approx 0.65$). Based on this effect size, with a significance level of 0.05 (two-tailed) and statistical power of 0.80, G*Power indicated a required sample size of 15 participants per group. Accordingly, a total of 30 individuals (15 PFPS, 15 controls) were included in the present study.

A cohort of young adult volunteers was recruited from local physiotherapy clinics in Hamadan, Iran for this cross-sectional study, including individuals diagnosed with patellofemoral pain syndrome (PFPS group) and a matched control group with no knee pain. The PFPS group consisted of 15 participants (9 females, 6 males; mean age 26.4 ± 4.8 years) with chronic anterior knee pain consistent with PFPS (duration >3 months). Key inclusion criteria for PFPS were reports of retropatellar or peripatellar pain exacerbated by at least two of the following activities: walking, running, stair ascent or descent, squatting, or prolonged sitting with knees flexed (Smith, 2012). Clinical diagnosis was confirmed by a sports medicine specialist; all PFPS subjects had positive findings on patellofemoral compression or Clarke's test and no history of patellar instability or ligament injury (Pereira Barbosa et al., 2024). Exclusion criteria included any history of knee ligament rupture, meniscal injury, fracture, or knee surgery, as well as any neurologic disorder or other lower-extremity injury in the past year (Yoon & Son, 2022). The control group comprised 15 healthy individuals (10 females, 5 males; mean age 25.7 ± 5.1 years) with no history of knee pain or injury, approximately comparable to the PFPS group in terms of age and activity level, though not fully matched for sex. Demographic characteristics of both groups are summarised in Table 1. All participants provided written informed consent, and the study protocol was approved by the Ethics Committee of Nahavand University (Approval Code: IR.NAHGU.REC.1399.009), adhering to the 1964 Declaration of Helsinki and its later amendments.

Experimental protocol. Participants walked at a self-selected comfortable speed along a 15 m walkway while electromyographic and foot-switch data were collected. After a brief warm-up, subjects performed practice trials to acclimate to the laboratory setting. During testing, each subject completed at least five walking trials; for each PFPS subject, the symptomatic limb was analysed (for bilateral cases, the self-reported more painful side was chosen), and for each control, a matched limb (right or left, selected to correspond to the side analysed in the PFPS counterpart) was used. Surface electromyography (EMG) electrodes (Ag/AgCl bipolar surface electrodes, 10 mm diameter, 20 mm inter-electrode distance) were placed over the following muscles of the test limb: vastus lateralis (VL) and rectus femoris (RF) – primary knee extensors; semitendinosus (ST) – a representative knee flexor (hamstring); tibialis anterior (TA) and extensor digitorum longus (ED) – ankle dorsiflexors; medial gastrocnemius (GM), lateral gastrocnemius (GL), and soleus (SO) – ankle plantar flexors (which also assist with knee flexion for the biarticular gastrocnemii). Electrode placement followed SENIAM guidelines for consistency (Majlesi et al., 2020), and skin preparation (shaving, light abrasion, alcohol cleaning) was performed to minimise impedance. A ground electrode was placed over the tibial shaft. Foot-switch pressure sensors were attached under the heel and forefoot to detect initial contact and toe-off events for gait phase iden-

tification. Participants were instructed to walk naturally and were not given a specific cadence or speed. Trials in which subjects visibly limped or altered their gait due to pain were discarded and repeated after rest. PFPS subjects rated their knee pain during walking on a 0–10 scale; mild to moderate pain (average ~3/10) was typically reported during the test, but none had to stop due to severe pain (Piva et al., 2009).

Data acquisition and processing. EMG signals were sampled at 1,000 Hz using a 16-channel wireless system (*Biomonitor ME6000, Megawin, Kuopio, Finland*), synchronised with foot-switch inputs. Raw EMG signals were band-pass filtered (20–450 Hz, 4th-order Butterworth), full-wave rectified, and then low-pass filtered at 6 Hz to create linear envelope profiles (Hajilou et al., 2024). Electromyographic data were normalised by dividing each muscle's signal by its peak amplitude within the corresponding gait cycle, allowing for within-cycle comparison of muscle activation patterns (Ortega-Auriol et al., 2025). Each gait cycle was defined from heel strike to the subsequent ipsilateral heel strike using foot-switch data. Gait cycle time was then normalised to 100%, and each EMG profile was interpolated to 101 time points (0%–100%) using cubic spline interpolation (Lamoth et al., 2006). For each participant, three representative gait cycles (consistent in timing and free of artifacts) were selected and ensemble-averaged to create a subject-level activation profile for each muscle. In addition to analysing individual muscle profiles, we computed group-level average profiles for synergistic muscle groups: quadriceps (mean of VL and RF) versus hamstrings (ST) at the knee, and plantar flexors (GM, GL, SO) versus dorsiflexors (TA, ED) at the ankle. For each muscle and each participant, we extracted peak activation magnitude and peak activation timing (as % of gait cycle) to compare between groups.

Co-contraction index (CCI): To quantify antagonist co-activation, we calculated a CCI for the knee and ankle joints using the processed EMG data (Besier et al., 2003). The knee CCI between quadriceps and hamstrings was computed at each percentage of gait cycle using the following formula:

$$CCI\_knee(t) = [2 \times \min(Q(t), H(t))] / [Q(t) + H(t)]$$

Where Q(t) is the normalised activation of the quadriceps group (mean of VL + RF) and H(t) is the activation of the hamstring (ST) at time t. The function min(Q(t), H(t)) returns the minimum of the two, reflecting overlapping activation. This index yields 0% when one muscle group is completely inactive and 100% when both are equally active. The ankle CCI between plantar flexors and dorsiflexors was computed similarly. This formulation follows the method originally described by Rudolph et al. (2000), who developed and validated a CCI to quantify simultaneous muscle activation during dynamic tasks such as hopping in ACL-deficient individuals (Rudolph et al., 2000).

Flexor–extensor pattern correlation: To assess the synchrony of muscle activation patterns between agonist and antagonist groups at the knee, Pearson correlation coefficients (r) were computed between the time-normalised (101-point) average EMG profiles of the knee flexor (ST) and the knee extensors (average of VL and RF) for each group (Van Can et al., 2024). This approach provides a global measure of in-phase (synchronous) or out-of-phase (reciprocal) activation timing across the gait cycle (Hautala et al., 2021). Higher r-values indicate greater similarity in the timing of signal fluctuations, while lower or negative values suggest more reciprocal patterns. Although individual-level activation trends were visually inspected for consistency, statistical comparison of individual correlation coefficients was not performed due to the descriptive nature of this analysis and limited sample size.

Statistical analysis. Statistical analyses were performed using MATLAB and SPSS software. For each muscle, independent-samples *t*-tests (two-tailed) were used to compare PFPS vs. control group means for the outcome variables of peak activation timing (% stance) and peak activation amplitude (%MVC). Levene's test was used to check homogeneity of variances; in cases of unequal variances, Welch's *t*-test was applied. Group differences in the average CCI for the knee and ankle were also assessed with *t*-tests. We adopted a significance level of $\alpha = 0.05$ for all comparisons. Additionally, we

reported the Pearson correlation coefficients describing flexor–extensor pattern similarity in each group. Results are reported as mean ± standard deviation unless otherwise noted.

3. RESULTS

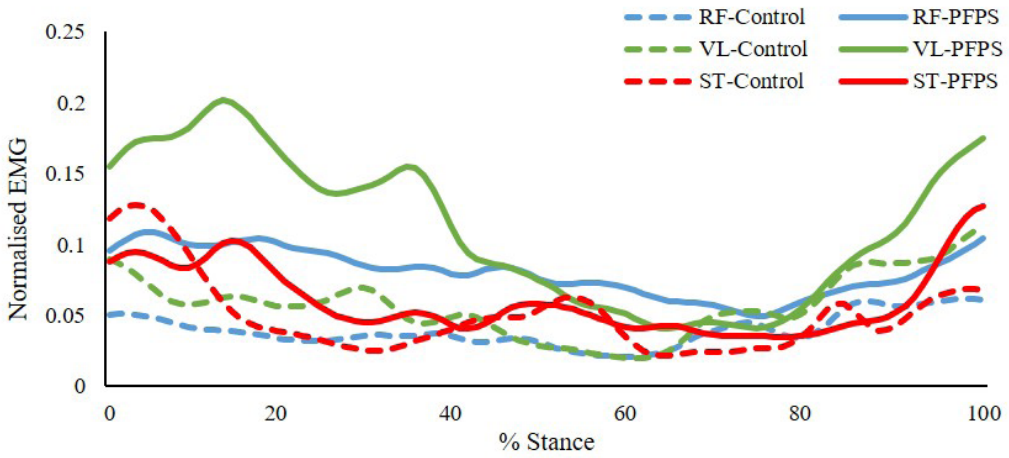
Table 1 presents the demographic characteristics of the study participants, showing that the PFPS and control groups were largely comparable across key variables such as age, height, weight, and BMI, with only minor differences in sex distribution.

Table 1. **Demographic characteristics of study participants**

	PFPS group (n = 15)	**Control group (n = 15)**	**p-value**
Age (years)	26.4 ± 4.8	25.7 ± 5.1	0.70
Sex (female / male)	9 / 6	10 / 5	0.73
Weight (kg)	72.4 ± 2.16	71.2 ± 5.86	0.47
Height (cm)	174.4 ± 7.51	176.2 ± 2.11	0.38
Body mass index (kg/m²)	23.8 ± 2.6	23.1 ± 2.9	0.49
Pain severity (daily life)	5.0 ± 1.5	0	–
Pain during walking (test)	2.8 ± 1.4	0	–

Note: p-values were calculated using independent samples *t*-tests. No statistical comparison was performed for pain severity and pain during walking because the control group consistently reported a value of zero.

Regarding muscle activation patterns, Fig. 1 illustrates the differences between the PFPS and control groups. Specifically, the PFPS group reached earlier quadriceps peaks (VL: 13% vs 100%, RF: 4% vs 98% of stance; both $p < 0.05$) with greater peak amplitudes (VL: 0.202 vs 0.112, RF: 0.109 vs 0.062; both $p < 0.05$). In contrast, the ST peak was significantly delayed (100% vs 3% of stance; $p < 0.05$) although its peak amplitude did not differ between groups (0.127 vs 0.128, $p = 0.78$).



Notes: Dashed lines = control mean; Solid lines = PFPS mean. Blue/Green = quadriceps (vastus lateralis, VL; rectus femoris, RF); Red = hamstring (semitendinosus, ST). PFPS exhibits higher quadriceps activation in early stance and a delayed hamstring peak compared to controls.

Figure 1. **Normalised EMG activation profiles of knee extensor (quadriceps) and flexor (hamstring) muscles during the gait cycle (stance phase)**

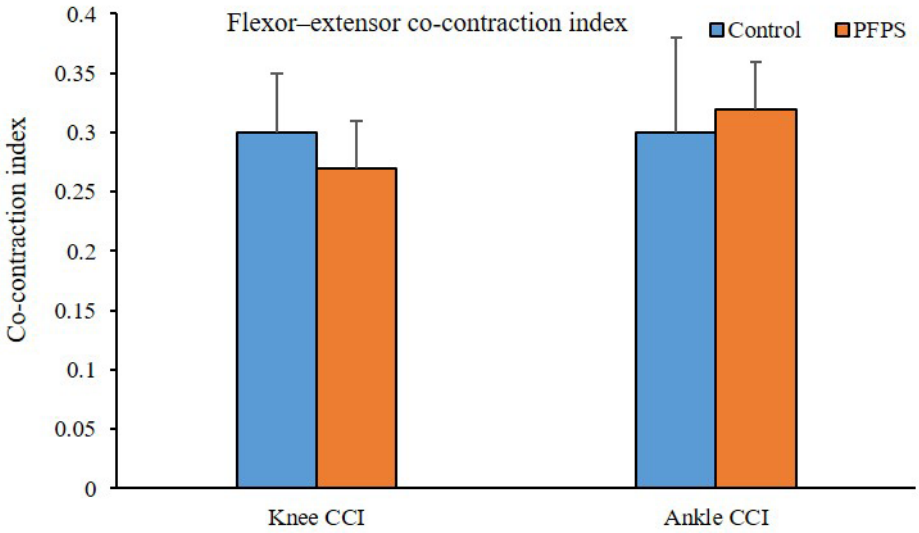
Peak activation timing and magnitude are further detailed in Table 2. The PFPS group demonstrated a higher GM peak amplitude (0.282 vs 0.161, $p < 0.05$) and TA peak timing (60% vs 100% of stance, $p < 0.05$) compared with controls. No significant group differences were found for the GL ($p = 0.41$), SO ($p = 0.36$), or ED ($p = 0.52$).

Table 2. **Per-muscle peak activation timing and amplitude (mean ± SD) for control and PFPS groups during stance**

Muscle	Peak timing (Control) (%)	Peak timing (PFPS) (%)	Peak amplitude (Control)	Peak amplitude (PFPS)	Timing p-value	Amplitude p-value
VL	100	13	0.112	0.202	**<0.05**	**<0.05**
RF	98	4	0.062	0.109	**<0.05**	**<0.05**
ST	3	100	0.128	0.127	**<0.05**	0.78
GM	2	9	0.161	0.282	0.32	**<0.05**
GL	40	42	0.281	0.24	0.67	0.56
SO	38	40	0.508	0.359	0.45	0.34
TA	100	60	0.27	0.242	**<0.05**	0.23
ED	56	56	0.219	0.182	0.89	0.44

Notes: P-values from independent t-tests (PFPS vs. Control). Significant differences ($p < 0.05$) are **bolded**.

Flexor–extensor co-contraction. Fig. 2 compares the flexor–extensor CCI between PFPS and control groups. Despite increased overlap in muscle activation patterns (higher synchrony measured by Pearson correlation: PFPS $r \approx 0.82$, control $r \approx 0.39$), no significant differences were observed in average CCI values at the knee ($p = 0.23$) or the ankle ($p = 0.34$).



Notes: Left = knee (quadriceps–hamstrings) CCI; Right = ankle (plantar flexor–dorsiflexor) CCI. Higher values indicate a greater proportion of simultaneous antagonist activation. No significant group differences were found in either case ($p > 0.2$).

Figure 2. **Comparison of flexor–extensor co-contraction index (CCI) between PFPS and control groups (mean ± SD)**

4. DISCUSSION

This study investigated differences in muscle activation during the gait cycle between individuals with PFPS and healthy controls. The key findings were: (1) individuals with PFPS exhibited significantly earlier and greater peak activation in the quadriceps muscles (VL, RF), along with delayed peak activation in the hamstrings (ST), indicating a temporal shift in knee muscle coordination; (2) this altered timing resulted in higher synchrony between agonist and antagonist muscles, as evidenced by greater Pearson correlation values. However, mean co-contraction indices (CCI) did not significantly differ between groups. This discrepancy may reflect the limitations of CCI as a global metric, which averages activation overlap across the gait cycle and may not capture subtle but clinically meaningful differences in timing and duration of muscle co-activation. PFPS participants appeared to exhibit more sustained, moderate overlap in muscle activity, whereas control participants showed shorter and more phase-specific bursts, suggesting different neuromuscular strategies not fully revealed by CCI alone; (3) PFPS participants also demonstrated altered ankle muscle patterns, including increased early peak activation of the GM and slightly reduced SO activity during terminal stance. These findings collectively suggest that PFPS is associated with a reorganisation of neuromuscular control strategies during walking, involving not only strength impairments but also disrupted timing and coordination across joints.

In PFPS participants, the quadriceps muscles reached peak activation within the first 15% of the gait cycle – earlier than typically observed in healthy individuals, whose peak generally occurs in mid- to late stance phases (Choi et al., 2025). This early peak may represent a compensatory strategy to stabilise the knee immediately after ground contact, possibly due to reduced shock absorption or gait stiffness, which are commonly reported in PFPS (Dierks et al., 2008). Such early and greater activation may also increase patellofemoral joint stress at a time of rising contact forces. This notion aligns with the modelling results of Sinclair et al. (2022), who found that runners with PFPS showed greater RF contributions to knee joint moments and impulse forces compared to controls, suggesting potential overuse of this muscle.

The ST muscle in PFPS showed delayed peak activity, typically occurring in mid- or late stance, whereas control participants demonstrated peak activity in early stance. Normally, hamstrings co-activate with quadriceps early in the gait cycle to stabilise the knee (Choi et al., 2025). This lack of early ST activation in PFPS may indicate altered motor control or pain-induced neuromuscular inhibition. While Choi et al. (2025) observed increased early hamstring activity in PFPS, discrepancies across studies may reflect subgroup differences, symptom severity, or task variations. In our study, late hamstring activation may serve to assist hip extension, possibly compensating for reduced ankle plantarflexor push-off.

Although flexor–extensor activation was more synchronised in PFPS (as indicated by higher Pearson correlation coefficients), group differences in average CCI were not statistically significant. One possible explanation is that traditional CCI metrics average values across the gait cycle, which may obscure dynamic shifts in coordination. In our data, PFPS participants appeared to exhibit prolonged, moderate co-activation, while controls had more discrete bursts. Even moderate but sustained co-activation may contribute to increased cumulative joint loading (Tsai et al., 2012), highlighting the importance of analysing activation timing alongside magnitude. This is consistent with Cowan et al. (2001), who reported altered neuromuscular coordination in PFPS without increased quadriceps amplitude.

At the ankle, PFPS participants exhibited significantly greater early medial gastrocnemius (GM) activation, which may support knee stabilisation as this muscle acts as both a knee flexor and ankle plantarflexor. This compensatory role could be a response to diminished early hamstring activation. On the other hand, reduced soleus activity may reflect a strategy to avoid excessive push-off force and thereby

minimise patellofemoral joint stress. These results are in line with findings in PFPS runners who demonstrated decreased ankle plantarflexion moments and power in late stance (Dierks et al., 2008).

Taken together, the observed pattern of earlier quadriceps and delayed hamstring activation in PFPS represents a phase shift in muscle coordination. This may lead to a stiffer, less energy-efficient gait, possibly increasing patellofemoral joint loading during walking or downhill movement. Although we did not measure the vastus medialis obliquus (VMO), previous research has demonstrated delayed VMO activation in PFPS (Cowan et al., 2001). A hypoactive VMO may force compensatory overreliance on VL and RF, exacerbating lateral patellar tracking and contributing to joint stress (Hug et al., 2015; Souissi et al., 2017). Clinically, these findings support the use of neuromuscular retraining interventions, not just strengthening programmes. Therapies targeting appropriate timing and coordination – such as biofeedback, motor control drills, and eccentric hamstring training – may restore more balanced muscle function. Given the increased GM activation observed, interventions addressing calf flexibility and kinetic chain alignment may also be beneficial.

This study has some limitations. First, EMG normalisation was based on peak values during gait rather than maximal voluntary contractions, which may reduce cross-subject comparability. Second, although three trials were analysed per participant, the use of averaged activation profiles may still mask some stride-to-stride variability. Third, we did not record the vastus medialis obliquus (VMO), and joint loading was not directly measured. Finally, all assessments and EMG electrode placements were performed by an experienced researcher in biomechanics who had formal training in surface EMG methodology and followed SENIAM guidelines to ensure reliability.

5. CONCLUSIONS AND PERSPECTIVES

Individuals with PFPS exhibit distinct neuromuscular alterations throughout the gait cycle, including earlier peak activation and greater amplitude in the quadriceps muscles (VL, RF), along with delayed activation of the hamstrings (ST). These phase-shifted patterns result in increased synchrony between agonist and antagonist muscles but do not necessarily translate to higher average co-contraction levels. Such abnormal timing may reflect a compensatory strategy to stabilise the knee but can increase cumulative patellofemoral joint stress. Importantly, altered activity was not limited to the knee but also involved ankle muscles, including increased early GM and reduced SO activation. These findings underscore the need for rehabilitation approaches that go beyond muscle strengthening. Clinical interventions should emphasise neuromuscular retraining to restore proper timing, particularly enhancing early hamstring activation and regulating quadriceps engagement. Gait retraining, motor control exercises, and addressing calf muscle dynamics should be integrated into comprehensive rehabilitation plans. Understanding how individuals with PFPS differ from healthy controls is clinically important because it helps identify specific neuromuscular deficits that contribute to pain and dysfunction, thereby guiding the design of targeted and more effective rehabilitation strategies. Promoting coordinated activation between knee and ankle muscles may help reduce joint loading, alleviate pain, and improve functional outcomes in PFPS patients.

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Lyginamoji asmenų, turinčių girnelės ir šlaunikaulio skausmo sindromą, apatinių galūnių raumenų aktyvacijos ir vienalaikio susitraukimo ėjimo metu analizė

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Santrauka

Tyrimo pagrindimas. Girnelės ir šlaunikaulio skausmo sindromas, arba patelofemoralinio skausmo sindromas (PFPS), yra dažna raumenų ir skeleto sistemą veikianti būklė, pasireiškianti priekinės kelio dalies skausmu ir apribojanti tokias kasdienes veiklas kaip vaikščiojimas ar lipimas laiptais. Vis daugiau tyrimų rodo, kad neuroraumeninės koordinacijos sutrikimai taip pat reikšmingai prisideda prie eisenos pakitimų PFPS atvejais.

Tikslas. Šio tyrimo tikslas – palyginti apatinių galūnių raumenų aktyvacijos laiką, intensyvumą ir vienalaikį susitraukimą tarp PFPS patiriančių asmenų ir sveikų kontrolinės grupės dalyvių ėjimo metu.

Metodai. Tyrime dalyvavo 15 PFPS pacientų ir 15 pagal amžių bei fizinį aktyvumą panašių sveikų asmenų. Einant pasirinktu tempu, buvo registruojami aštuonių apatinių galūnių raumenų elektromiografijos (EMG) rodikliai. Duomenys apdoroti siekiant nustatyti didžiausios aktyvacijos laiką bei kelio ir čiurnos raumenų vienalaikį susitraukimą. Grupių skirtumai analizuoti naudojant *t* testus ir Pearsono koreliacijos koeficientą.

Rezultatai. PFPS grupėje nustatyta ankstyvesnė (*VL*: 13 % ir 100 %; *RF*: 4 % ir 98 % atramos fazės, $p < 0,05$) ir didesnė (*VL*: 0,202 ir 0,112; *RF*: 0,109 ir 0,062, $p < 0,05$) keturgalvio raumens aktyvacija, vėlyvesnė (*ST*: 100 % ir 3 % atramos fazės, $p < 0,05$) dvigalvio šlaunies raumens aktyvacija bei didesnė dvilypio blauzdos raumens vidinės dalies aktyvacija (*GM*: 0,282 ir 0,161, $p < 0,05$). Nors raumenų vienalaikio susitraukimo reikšmės tarp grupių reikšmingai nesiskyrė, PFPS grupėje nustatytas žymiai didesnis agonistų ir antagonistų vienalaikiškumas ($r \approx 0,82$, palyginti su $r \approx 0,39$ kontrolinėje grupėje).

Išvados. PFPS patiriantiems asmenims ėjimo metu būdingi neuroraumeninės kontrolės pakitimai, įskaitant tiek kelio, tiek čiurnos raumenų aktyvacijos laiko pokyčius. Šie pokyčiai gali padidinti sąnarių įtampą ir lemti ilgalaikį skausmą. Siekiant sumažinti skausmą ėjimo metu, reabilitacija turėtų būti orientuota ne tik į jėgos stiprinimą, bet ir į raumenų aktyvacijos laiko ir koordinacijos atkūrimą.

Reikšminiai žodžiai: patelofemoralinio skausmo sindromas; ėjimas; elektromiografija; raumenų aktyvacija; vienalaikis susitraukimas

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