scientific reports

Check for updates

OPEN

Lower extremity electromyographic characteristics of patients with noncontact anterior cruciate ligament rupture in one-legged jump landings: a case-control study

Jie Xu[1], Meng Chen[2], Jing Ran[1] & Xiaobing Luo[1]✉

There have been many studies on neuromuscular adaptation after anterior cruciate ligament (ACL) reconstruction, while the understanding of muscle activation patterns in unreconstructed patients with ACL rupture is still limited. The aim of this study was to investigate the lower limb electromyographic characteristics of unreconstructed patients with complete ACL rupture in a single-legged hopping landing task in order to deepen the understanding of motor control strategies in the ACL-deficient state and to provide a reference for rehabilitation assessment and intervention. Forty-two subjects were recruited for this study using a case-control design, with an ACL injury group ($n = 21$) of patients with unilateral non-contact complete rupture without reconstruction and a control group ($n = 21$) of healthy individuals matched for gender, dominant leg, and level of exercise. All subjects completed a single-leg hop landing task and synchronized Noraxon Ultium surface EMG signals with Bertec force plate data via the QUALISYS 3D motion capture system. EMG data were recorded from the lateral femoral (VL), medial femoral (VM), biceps femoris (BF), semitendinosus (ST), and gluteus maximus (Gmax) muscles before and after the landing for 100 ms each. Calculated metrics included activation onset time (onset-IC), peak appearance time (peak-IC), activation duration, and standardized root mean square (RMS) values. Data were analyzed by two-way ANOVA or nonparametric Scheirer-Ray-Hare test, and the significance level was set at $p < 0.05$. BF ($p = 0.0409$) and Gmax ($p = 0.0469$) sustained activation of the dominant leg in the injury group was significantly longer than that of the dominant leg in the control group. The onset-IC of BF ($p = 0.0457$), ST ($p = 0.0277$), and Gmax ($p = 0.0192$) of the dominant leg in the injury group was significantly earlier than that of the dominant leg in the control group. The peak-IC of BF ($p = 0.0457$) and ST ($p = 0.0280$) of the dominant leg in the injury group was significantly later than that of the dominant leg in the control group. The peak RMS of VL ($p = 0.0171$), VM ($p = 0.0054$), and Gmax ($p = 0.0003$) in the dominant leg of the injury group was significantly lower than that of the dominant leg of the control group in 100 ms after IC. Unreconstructed patients, averaging 18 months after ACL injury, continued to maintain a similar muscle pre-activation sequence as healthy individuals during the jump landing task, but showed a prolonged activation duration and reduced activation intensity, suggesting that neuromuscular activity was adjusted to maintain the kinematic profile. The delay in the peak of the posterior muscle groups (especially BF and ST) may be used to synergize tibial rearward movement and reduce forward movement and internal rotation, thus constituting a compensatory protective mechanism. The results of this study provide evidence for neuromuscular adaptation in the ACL-deficient state and are informative for preoperative functional assessment and rehabilitation intervention strategies.

Keywords ACL, Noncontact injury, Surface electromyography, Rehabilitation, Isometric muscle strength, Root mean square amplitude

[1]Department of Sports Medicine, Sichuan Provincial Orthopedics Hospital, Chengdu, China. [2]Department of Emergency Medicine, Nanchong Hospital of Traditional Chinese Medicine, Nanchong, China. ✉email: 202230470060@mail.scut.edu.cn

Anterior cruciate ligament (ACL) injury is one of the common knee injuries in clinical practice, which seriously affects the athletic ability and knee stability of patients. According to statistics, ACL rupture occurs in about 250,000 people per year in the United States[1], the incidence rate in Sweden is about 78–81 cases/100,000 people/year[2], and the rate of ACL injury in Chinese national-level athletes is about 0.47%[3]. The incidence of ACL injuries and reconstruction is on the rise as the percentage of youth participating in competitive sports increases, and as middle-aged and older adults remain physically active for longer periods of time. ACL reconstruction has become the primary treatment for this injury. Annual expenditures in the United States for ACL reconstruction and rehabilitation reach $7.6 billion and $17.7 billion, respectively, highlighting the significant burden of disease associated with this injury[4]. Furthermore, the risk of re-injury following ACL reconstruction remains significant. Studies indicate that after the initial reconstruction, the risk of rupture in the unaffected knee is even higher than the risk of re-rupture in the injured knee[5,6]. Surgical treatment can restore the physiological structure and function of the patient's knee joint, but systematic rehabilitation training is still required to regain daily living and athletic abilities. In terms of injury mechanisms, non-contact ACL ruptures predominantly occur during high-dynamic movement scenarios such as landing from a jump or sudden stops and changes of direction[7], accounting for approximately 72%–95% of all ACL ruptures[8].

Current surface electromyography studies on neuromuscular activity following ACL injury predominantly focus on major motor muscle groups such as the quadriceps femoris, hamstrings, and triceps surae[9–11]. Existing research indicates that insufficient gluteal muscle control can lead to dynamic knee varus, thereby increasing the risk of non-contact ACL injuries[12]. Therefore, investigating the role of the Gmax in dynamic knee control is crucial. Furthermore, most studies in this field employ relatively low-impact functional tasks such as walking[13], running[14], and squatting[15] for EMG analysis. Given that the most common ACL injury movements are jumping and landing, emergency deceleration, and lateral changes of direction, in order to reproduce muscle EMG data closer to the actual moment of injury, the test movement needs to be as consistent as possible with the injury movement. In addition, most of the research subjects in this field are individuals who have undergone ACL reconstruction surgery, and there is a lack of research on the muscle activation mechanism of patients without reconstruction during high-dynamic movements.

Since ACL injuries are typically not caused by direct external force but are closely related to an individual's inherent movement patterns, they are considered a type of injury that can be prevented through intervention. This underscores the critical importance of clearly identifying the differences in electromyographic timing and intensity characteristics of lower limb muscle groups between ACL rupture patients and healthy individuals. Simultaneously, to prevent recurrent injuries to the unaffected and affected side in ACL-injured patients, targeted rehabilitation interventions based on the characteristics of electromyographic timing and intensity changes in the lower limb muscle groups of both the affected and unaffected sides following ACL rupture are crucial. Based on the aforementioned research gaps, this study targets patients with chronic non-contact ACL complete tears who have not undergone reconstruction. It utilizes a single-leg jump landing task to analyze the activation timing, activation duration, and activation intensity characteristics of key muscle groups, including the vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), semitendinosus (ST), and gluteus maximus (Gmax), in both the injured and uninjured limbs, and compares them with corresponding limbs in the control group. Aims to provide a theoretical foundation for guiding targeted preoperative rehabilitation interventions for ACL rupture patients and early ACL injury prevention for sports enthusiasts.

Materials and methods

Participant recruitment

The study used G*Power (version 3.1; https://www.psychologie.hhu.de/arbeitsgruppen/allgemeine-psychologie-und-arbeitspsychologie/gpower) software for sample size estimation. The level of significance was set at $\alpha = 0.05$, and the target statistical power was 80%. Based on Cohen's recommendation[16], the effect size (ES) was set at 0.46, and the minimum required sample size was calculated to be 40 individuals (20 in each group) using a two-way ANOVA model. Considering a dropout rate of approximately 10%, the final plan was to include 22 subjects in each group, for a total of 44, to ensure adequate statistical efficacy. All patients with ACL injuries completed testing before undergoing ACL reconstruction. Healthy individuals of the same sex, dominant leg side, and similar level of exercise (Tegner score not differing by more than ± 1 point) were screened by questionnaire to serve as the control group. All enrolled patients received no systematic physical therapy or rehabilitation intervention post-injury. They underwent standardized rehabilitation training at the Knee Sports Injury Department of Sichuan Orthopedic Hospital for three days following admission and prior to surgery. Detailed exercise prescriptions are provided in Supplementary Table 1. The control group received the same volume of exercise training. Considering that adaptive changes occur in the neuromuscular system of patients more than six months after ACL injury, indicating that the neuromuscular reorganization process following ACL injury has reached a certain stage, this allows us to observe the significant impact of ACL injury on electromyographic activity in lower limb muscle groups[17]. Therefore, we selected a disease duration criterion of more than six months for complete ACL injury. All participants signed informed consent forms prior to the experiment. All methods were carried out in accordance with relevant guidelines and regulations, including the

Good Clinical Practice (GCP) guidelines and the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of Sichuan Orthopedic Hospital (Ethics No. KY2020-017-01). The inclusion criteria were: injury group: (1) age 18–35 years old; (2) mode of injury was non-contact injury; (3) the patient's injured leg was his/her dominant leg. We have determined that the patient's dominant leg did not change before or after the injury (the dominant leg is defined as the leg preferred for kicking, standing, jumping, or stepping on a box); (4) MRI showed a complete rupture of the ACL alone; (5) the duration of the disease was more than six months; (6) there was no injury to the adjacent joints; and (7) there was no underlying disease such as gout or rheumatoid arthritis. Control group: (1) Age: 18–35 years old, regardless of gender; (2) No motor system diseases in the last six months; (3) No history of lower limb surgery.

Data collection

In this study, a QUALISYS 3D motion capture system (Model Oqus 300, Sweden) was synchronized with a Noraxon wireless surface EMG system (Model Ultium EMG, sampling frequency 2000 Hz, USA) and a Bertec force table (Model FP4060-08, sampling frequency 200 Hz, USA) for motion data acquisition. The force table was used to detect the initial contact (IC) time, defined as the instant when the vertical ground reaction force first exceeded 10N[18]. EMG acquisition sites were referenced to the SENIAM standardized localization guidelines[19] as detailed in Supplementary Table 2. Subjects underwent a 10-minute warm-up prior to testing, consisting of 5 min of jogging with 5 min of dynamic stretching. After warming up, the area to which the electrodes were attached was prepared by cleaning with 75% alcohol, shaving, exfoliating with fine sandpaper, and again cleaning with an alcohol cotton ball and air-drying to minimize skin surface impedance. Subsequently, silver/silver chloride electrodes (model CH3236, CATHAY, China) were affixed with an electrode spacing of 20 mm[20], the reference electrode was kept equidistant from the acquisition electrode and secured against dislodgement by means of a muscle patch with medical adhesive tape (see Fig. 1)[21]. In my test procedure, the one-legged jump landing task was given first priority, followed by the maximal voluntary contraction (MVC) test, with a 5-minute rest period between the two. Subjects were asked to stand with their feet shoulder-width apart on a 30 cm raised platform, with the distance between the platform and the center of the force plate set at 80% of the distance from the subject's anterior superior iliac spine to the medial ankle[22]. The EMG signals were recorded before the start of the jump, and the subjects maintained a one-legged standing position on command, stabilized and jumped forward without initial velocity, and remained stationary for 3 s after landing on one foot. Each subject completed 5 tests at 1-minute intervals. Visual Analogue Scale (VAS) was used during testing to assess subjects' pain levels while performing the single-leg landing task, with no significant discomfort observed throughout the process (see Fig. 2). Maximum voluntary contraction (MVC) testing was performed using the David isometric device, which measures the maximum flexion and extension forces of the anterior and posterior thigh muscle groups, respectively. To minimize interference with EMG results caused by variations in the distance between the instrument and the muscles, we adjusted the instrument's rotational axis according to the subject's seated position during measurement, aligning it with the greater trochanter of the femur on the outside of the knee joint being tested. The upper fixture held the thigh as close to the chair surface as possible. Furthermore, the slider was fixed 3 cm above the ankle joint, depending on the patient's leg length, to ensure maximum strength for each subject. For the Gmax MVC test, the subject was lying prone on a yoga mat with the legs abducted about 30°, the knees flexed 90°, and the hip joint in 0° extension. The experimenter applied resistance to the lower and middle thighs with his bare hands, and the subject tried his best to complete the hip extension and abduction movements under the oral command, with the knee joint slightly off the ground (about 2–3 cm) and holding it for 5 seconds[23]. All MVC tests were repeated 3 times at 30-second intervals with strong verbal encouragement to elicit maximal force output[24]. The MVC tests for the BF and ST muscles are shown in Fig. 3.

Data processing

VL, VM, BF, ST, Gmax muscle activation timing metrics

The raw surface electromyography (SEMG) signals were first visually inspected to ensure that the acquired data were complete and valid. Subsequently, the DC offset was removed using Noraxon EMG (Ultium version; https://www.noraxon.com) software and a fourth-order zero-delay Butterworth high-pass filter (cutoff frequency fc

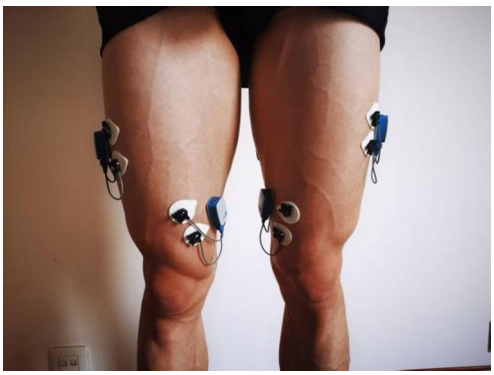

Fig. 1. VL, VM surface EMG acquisition site.

Fig. 2. One-legged jump landing test.

Fig. 3. MVC test diagram for BF and ST.

	Injury group ($N=21$)	Control group ($N=21$)	t-value	*p*-value
Gender (M/F)	11/10	11/10		
Age (y)	26.97 ± 4.35	24.66 ± 3.65	1.861	0.0702
Height (cm)	170.80 ± 10.92	172.40 ± 4.85	0.6298	0.5324
Weight (kg)	68.03 ± 12.25	72.47 ± 8.43	1.366	0.1794
BMI (kg/m2)	22.67 ± 2.94	23.92 ± 2.65	1.449	0.1552
Tegner Rating	5.14 ± 1.35	5.29 ± 1.3	0.3478	0.7298
Duration of injury (months)	18.20 ± 7.23			

Table 1. Basic information of subjects.

Muscle	Injury group		Control group		*p*-valuea
	Dominant leg (Affected limb)	Non-dominant leg (Healthy limbs)	Dominant leg	Non-dominant leg	
VL	354.12 ± 101.82	339.37 ± 84.57	337.64 ± 87.35	329.28 ± 105.18	0.8781
VM	336.45 ± 77.18	322.16 ± 80.08	311.22 ± 82.60	321.59 ± 86.27	0.4906
BF	346.61 ± 73.52	335.16 ± 65.84	305.52 ± 55.67*	315.43 ± 59.81	0.4472
ST	347.34 ± 66.86	338.24 ± 65.05	311.34 ± 57.10	319.97 ± 54.78	0.5084
Gmax	261.39 ± 52.60	257.49 ± 49.75	230.04 ± 42.81*	235.21 ± 55.30	0.6808

Table 2. Duration of sustained activation of each muscle comparison (ms). Note: [a], indicates the effect of subject group× test limb interactions on the muscle sustained activation time; *, indicates $p < 0.05$ for comparison (a), indicates the effect of subject group test limb interactions on the muscle sustained activation time; *, indicates $p < 0.05$ for comparison between the dominant leg in the injury group and the other groups.

	Muscle	Injury group		Control group		p-valuea
		Dominant leg (Affected limb)	Non-dominant leg (Healthy limbs)	Dominant leg	Non-dominant leg	
onset-IC	VL	-125.37b±45.43	-118.35±42.95	-109.62±43.79	-114.26±41.25	0.5398
	VM	-120.84±37.17	-114.66±35.49	-109.57±30.27	-103.36±34.34	0.9985
	BF	-150.55±66.04	-136.83±63.47	-112.86±55.67*	-129.28±54.67	0.2544
	ST	-156.59±66.93	-117.49±59.10*	-114.49±60.71*	-120.41±56.07	0.0938
	Gmax	-95.61±20.02	-87.79±17.67	-82.16±18.36*	-85.21±16.77	0.1760
peak-IC	VL	151.26±46.87	161.37±48.72	148.41±52.06	133.63±57.14	0.2701
	VM	146.64±59.81	148.22±65.44	157.80±74.42	160.71±70.32	0.9643
	BF	-34.21±12.22	-40.31±16.74	-42.83±10.38*	-37.25±13.11	0.0578
	ST	-32.15±12.46	-38.14±13.78	-40.36±11.33*	-38.34±9.59	0.1267
	Gmax	41.47±12.04	39.26±12.44	40.08±10.37	37.82±12.20	0.9924

Table 3. Comparison of onset-IC and peak-IC by muscle (ms) Note: [a], indicates the effect of subject groupx test limb interactions on the onset-IC and peak-IC time of muscle; [b], negative values indicate that they occurred before initial contact (IC); *, indicates $p<0.05$ for comparison of the dominant leg in the injury group versus the other groups.

= 15 Hz) was applied to remove motion artifacts. To extract temporal features of muscle activation, the filtered signal was full-wave rectified and a low-pass filter (fc = 20 Hz) was applied to generate a linear envelope signal. By experimenting with cutoff frequencies in the range of 10–25 Hz (in steps of 1 Hz), 20 Hz was finally selected as the low-pass filtering threshold, a parameter that maximizes the retention of key features consistent with changes in muscle tone[25]. The identification of muscle activation timing was based on a threshold of 15% of the maximum amplitude of the linear envelope signal: when 28 consecutive sampling points (sampling frequency 2000 Hz) exceeded or fell below this threshold, respectively, they were considered as the beginning and the end of muscle activation[26]. To determine this threshold, we conducted multiple rounds of comparisons and manual calibration within the range of 3%–25%. Specifically, we manually labeled the start and end times of each activation event while testing different threshold settings, comparing the automatically extracted activation sequences with the manually calibrated ones. Through repeated threshold adjustments, we evaluated the accuracy of muscle activation timing at each threshold. Ultimately, 15% was selected as the optimal threshold, as it accurately distinguished genuine muscle activation from background noise and aligned with prior research findings[27]. During manual calibration, we paid particular attention to how threshold selection affected signal clarity. Too low a threshold caused excessive background noise to be mistaken for muscle activation, while too high a threshold risked missing weaker muscle activation signals. The final determination of 15% as the optimal threshold accurately captured the onset and offset of muscle activation while minimizing noise interference. The final extracted temporal metrics included: the duration of muscle activation (ms); the time difference of activation onset time relative to initial contact (IC) (onset-IC, ms); and the time difference of peak EMG appearance time relative to IC (peak-IC, ms).

VL, VM, BF, ST, Gmax muscle activation strength indicators

Raw SEMG signals were full-wave rectified, smoothed using a moving-window (50 ms) root mean square (RMS) algorithm, and normalized by the peak value recorded during MVC. Muscle activation intensity indices were divided into the following two categories: preparatory muscle activity: peak and mean RMS values within a 100 ms window before IC; reactive muscle activity: peak and mean RMS values within a 100 ms window after IC.

Statistical analysis

Raw data were entered and organized using Microsoft Excel, and statistical analysis was done using GraphPad Prism (version 9.5; https://www.graphpad.com) software. All continuous variables were first assessed for normality by the Shapiro-Wilk test, and Levene's test for chi-square was used. For variables that satisfied both normal distribution and chi-square, a two-way ANOVA was used, with "group" (ACL injury group vs. control group) as a between-group factor and "limb side" (dominant vs. nondominant leg) as a within-group factor. If main effects or interactions were significant, post hoc two-by-two comparisons were further performed using Fisher's LSD method. Given the small sample size of this study, employing Fisher's LSD method as the post-hoc comparison technique is a reasonable choice. This method is well-suited for multiple comparisons in small samples and effectively identifies intergroup differences. Although Fisher's LSD does not perform p-value correction, we minimized the risk of Type I errors by rigorously verifying all assumptions, including ensuring data normality. We did not employ Bonferroni correction because this method significantly reduces statistical power, particularly in multiple comparisons. It may lead to overly stringent significance levels, thereby increasing the risk of Type II errors (false negatives). Therefore, given the exploratory nature of this study and the limitations of its sample size, we consider a certain degree of Type I error acceptable. This approach helps maintain the sensitivity and statistical power of the analysis. All data are presented as mean ± standard deviation and the significance level was set at $p < 0.05$.

Results

Baseline characteristics of participants

During the testing process, one subject in the injury group withdrew from the trial because she gave up ACL reconstruction surgery and chose conservative treatment; one subject in the control group was excluded because she was unable to complete all the tests due to scheduling conflicts. In the end, a total of 42 subjects completed all the tests, including 21 in the injury group and 21 in the control group. Among them, 22 were male and 20 were female. The basic information of the subjects is shown in Table 1.

Muscle activation duration

Since no significant interaction effect was presented between subject group and test limb, the main effects of the group factor and limb factor were analyzed separately, and the results are shown in Table 2. In terms of muscle activation duration, subject group showed a significant main effect on BF ($F_{(1,40)} = 4.732$, $p = 0.0326$), with the duration of activation of the dominant leg in the injury group being significantly longer than that of the dominant leg in the control group ($p = 0.0409$). A significant main effect was also shown for Gmax ($F_{(1,40)} = 5.961$, $p = 0.0168$), which likewise showed a significantly longer duration of sustained activation of the dominant leg in the injury group ($p = 0.0469$).

Characteristics of EMG activation timing by muscle

Since there was no significant interaction effect between subject group and test limb, the main effects of the two factors were analyzed separately in this section, and the results are shown in Table 3; Fig. 4. In the comparison of onset-IC, the subject group showed a significant main effect on BF ($F_{(1,40)} = 5.127$, $p = 0.0351$), with onset-IC for the dominant leg in the injury group significantly earlier than that for the control group's dominant leg ($p = 0.0457$).ST also showed a significant main effect ($F_{(1,40)} = 6.314$, $p = 0.0239$), with the onset-IC of the dominant leg in the injury group being significantly earlier than that of the dominant leg in the control group ($p = 0.0277$) and that of its own non-dominant leg ($p = 0.0405$). In addition, Gmax also showed a significant main effect ($F_{(1,40)} = 4.053$, $p = 0.0474$), as evidenced by a significantly earlier onset-IC in the dominant leg of the injury group than in the dominant leg of the control group ($p = 0.0192$). In the comparison of peak-IC, BF showed a significant main effect ($F_{(1,40)} = 4.040$, $p = 0.0415$), in which the peak-IC of the dominant leg in the injury group was significantly later than that of the dominant leg in the control group ($p = 0.0457$).ST also showed a significant main effect ($F_{(1,40)} = 7.271$, $p = 0.0190$), with the dominant leg in the injury group having the peak-IC significantly later than the control dominant leg ($p = 0.0280$).

Characterization of EMG activation intensity by muscle

For the RMS peak versus mean within 100 ms before and after IC, there was no significant interaction effect between subject group and test limb, so the main effect of subject group versus test limb was analyzed, as shown in Figs. 5 and 6. Specific p values for the multiple comparisons results are detailed in Supplementary Table 3. There was a significant main effect of subject group on the peak RMS of the VL within 100 ms after IC ($F_{(1,40)} = 4.742$, $p = 0.0324$), and the dominant leg in the injury group was significantly lower than the dominant leg in the control group ($p = 0.0171$). There was a significant main effect of subject group on the peak RMS of VM within 100 ms after IC ($F_{(1,40)} = 6.007$, $p = 0.0164$), with the dominant leg in the injury group being significantly lower than the dominant leg in the control group ($p = 0.0054$) and the non-dominant leg in the control group ($p = 0.0253$). There was a significant main effect of subject group on the mean RMS of VM within 100 ms after IC ($F_{(1,40)} = 6.925$, $p = 0.0102$), which was significantly lower for the dominant leg in the injury group ($p = 0.0178$) and the nondominant leg in the injury group ($p = 0.0357$) than for the dominant leg in the control group. There was a significant main effect of subject group on the peak RMS of ST within the first 100 ms of IC ($F_{(1,40)} = 6.429$, $p = 0.0132$), which was significantly higher in the dominant leg of the injury group than in the dominant leg of the control group ($p = 0.0439$) and the non-dominant leg of the control group ($p = 0.0229$). There was a significant main effect of subject group on the mean RMS of ST within the first 100 ms of IC ($F_{(1,40)} = 5.563$, $p = 0.0208$), and the dominant leg was significantly higher in the injury group than the dominant leg in the control group ($p = 0.0483$). There was a significant main effect of subject group on the peak RMS of Gmax within 100 ms after IC ($F_{(1,40)} = 13.94$, $p = 0.0004$), with the dominant leg in the injury group being significantly lower than the dominant leg in the control group ($p = 0.0003$) and the nondominant leg in the control group ($p = 0.0122$), and the nondominant leg in the injury group being significantly lower than the dominant leg in the control group ($p = 0.0081$).

Discussion

ACL disruption is thought to disrupt pre-existing neuromuscular control strategies in the lower extremity. It has been shown that ACL injury can cause significant alterations in the kinematic, kinetic, and electromyographic characteristics of the lower extremity during high-impact tasks such as jump landings[28–30], and that these changes reflect a phenomenon of neuromuscular adaptation or reprogramming in response to joint instability aimed at enhancing the dynamic stability of the knee and reducing the risk of re-injury by modulating the order of activation, duration, and peak response. In the present study, we found that muscle activation durations were generally prolonged in the affected leg of the ACL-injured group during a single-leg jump landing task, with BF and Gmax being the most significant. This result is consistent with previous findings regarding prolonged EMG durations in walking, such that the VM, VL, ST, and lateral popliteus showed similar trends[31,32]. The prolonged activation duration may represent a compensatory strategy for knee instability, increasing joint compressive forces by maintaining longer muscle contractions, thereby enhancing mechanical stability and compensating for limitations caused by ACL deficits, resulting in a kinematic profile similar to that of the healthy side.

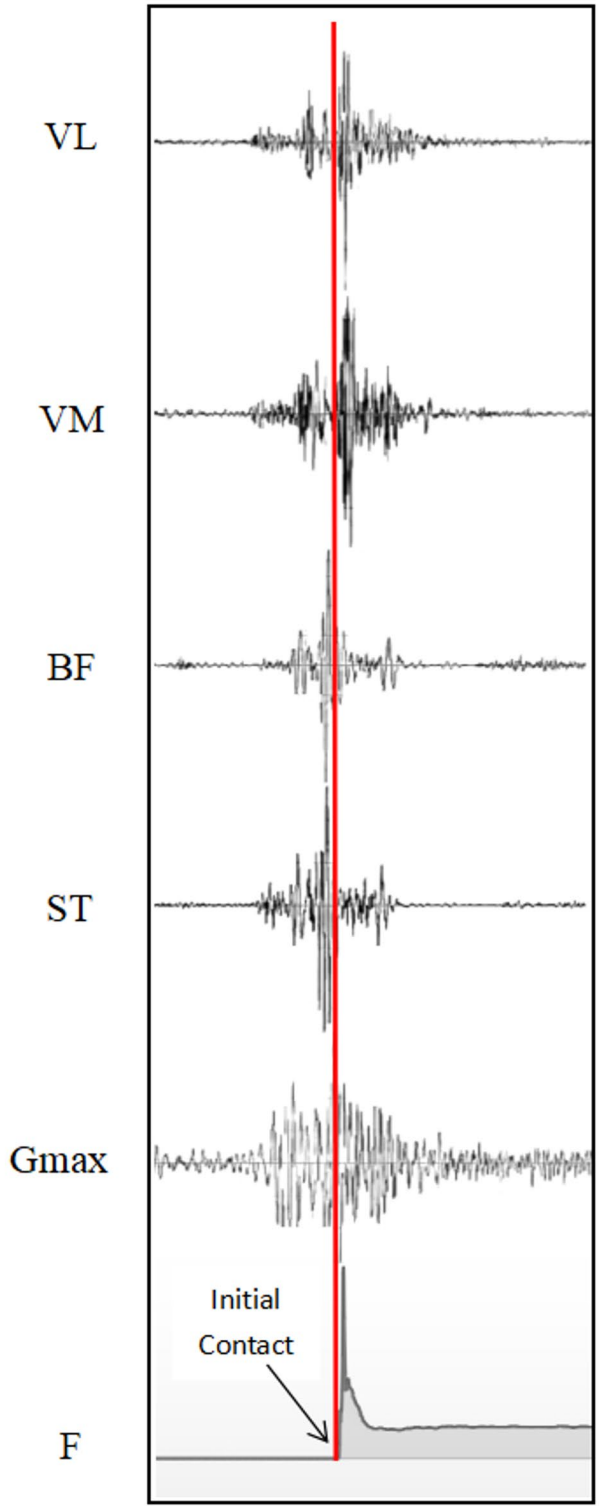

Fig. 4. Pre-activation sequence of each muscle before touchdown.

In terms of muscle activation timing, the present study observed that the onset of activation (onset-IC) of several key muscle groups in the ACL-injured group was significantly earlier than that of the control group, especially the posterior masseter muscles (BF, ST) and Gmax. This early pre-activation pattern was considered as part of the "pretension conditioning" during landing, which aims to By preactivating the muscles a sufficiently stable base prior to contact with the ground to control knee trajectory and absorb ground reaction forces. In the case of the quadriceps muscle, for example, it has been shown that healthy individuals preactivate the VM and VL 103–114 ms before ground contact, and the injury group in this study showed a similar or even earlier activation

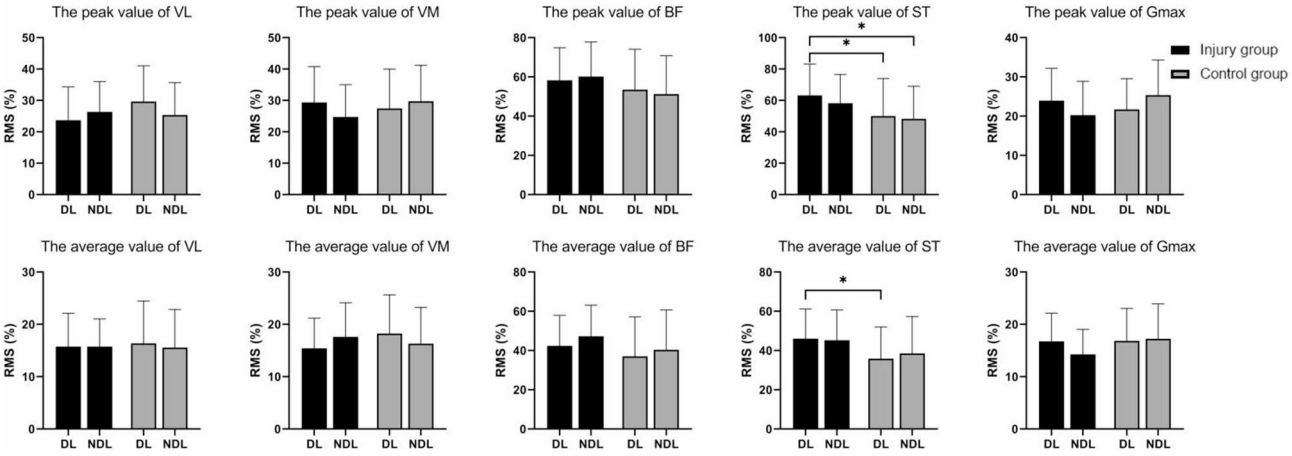

Fig. 5. Comparison of RMS in each muscle during the first 100 ms of IC. note: DL indicates dominant leg; NDL, nondominant leg; *, indicates $p < 0.05$ for comparison between the dominant leg in the injury group and the other groups; RMS, root-mean-square amplitude.

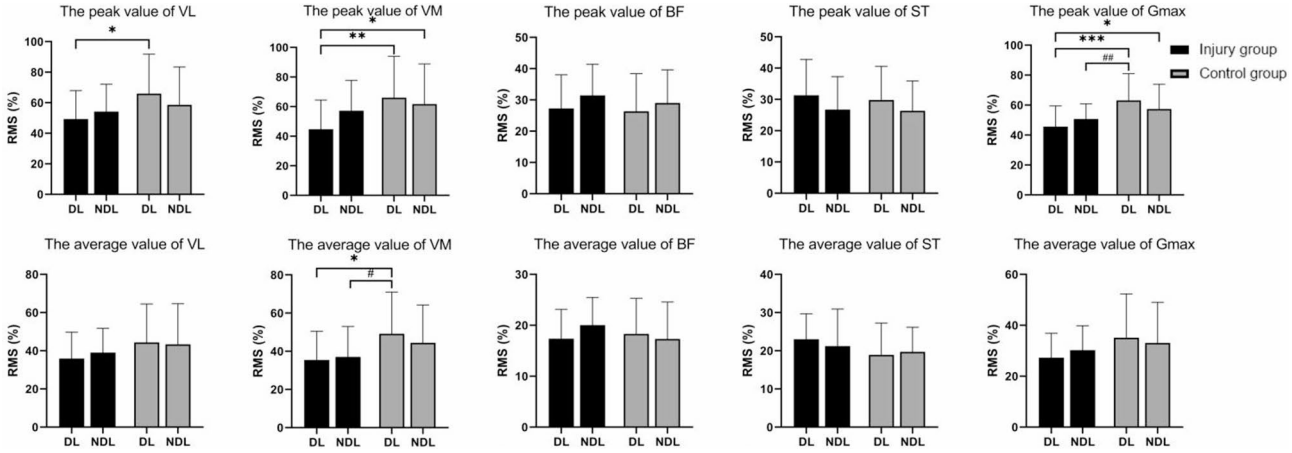

Fig. 6. Comparison of RMS of each muscle within 100ms after IC. note: DL indicates dominant leg; NDL indicates nondominant leg; *, indicates $p < 0.05$ for comparison between the dominant leg and other groups in the injury group; **, indicates $p < 0.01$ for comparison between the dominant leg and other groups in the injury group; ***, indicates $p < 0.001$ for comparison between the dominant leg and other groups in the injury group; #, indicates $p < 0.05$ for comparison between the nondominant leg and other groups in the injury group; ##, indicates $p < 0.01$ for comparison between the nondominant leg and other groups in the injury group; RMS, indicates root mean square amplitude.

sequence. In addition, there is an electromechanical delay (EMD) between the EMG signal and actual tension generation, which ranges from 20 to 100 ms for knee extensors[33] and may reach 55–92 ms for hamstrings[34], making early activation particularly critical. Analysis of the time of peak EMG appearance (peak-IC) revealed that both BF and ST peaks were significantly delayed in the ACL injury group, which is highly consistent with the hamstring function of action. The hamstrings are the main co-stabilizing muscles of the ACL by generating tibial "posterior drawer force" to reduce anterior displacement. During landing maneuvers, peak tibiofemoral shear occurs approximately 28–30 ms after touchdown[25,26]. Therefore, a delay in maximal hamstring output to this stage coincides with the most vulnerable window of the ACL, thus forming a dynamic protective barrier. Blackburn et al.[34] state that hamstring EMD is 72 ms, which is highly consistent with the result that hamstrings reach peak activity approximately 30–40 ms before touchdown. This is highly consistent with the result that the hamstrings reach peak activity around 30–40 ms before touchdown.

Gmax showed a similar pattern of regulation. Not only was Gmax activated earlier before touchdown in the ACL injury group, its peak activation also occurred around 40 ms after landing, and the abduction moment generated by Gmax was expected to peak 92 ms after landing. This coincides with the electromechanical delay (52 ms on average) in hip abduction proposed by Kim et al.[35], suggesting that its abduction output is functioning right at the peak tibiofemoral shear force phase. It has been reported that the peak knee valgus angle mostly occurs 85 ms after landing (range 70–100 ms), and more than 60% of athletes reach their maximal knee valgus

angle at this stage[36,37]. Therefore, early activation of Gmax helps to counteract the tendency of hip adduction and knee valgus and prevents pelvic instability from adversely affecting the knee joint. Overall, patients with ACL injuries show a typical "pre-activation-post-peak" bi-directional strategy in landing tasks: key muscle groups are activated early to pre-establish muscle tone in response to ground impact, and the peak is moderately delayed to match the timing of the peak of the ground reaction force and the emergence of shear forces, thus maximizing their ability to respond to ground impact. This reprogramming of neuromuscular timing is clinically associated with reduced reinjury risk, improved motor control, and enhanced patient functional scores. Therefore, targeted strengthening of the preactivation and timing coordination of the hamstrings and Gmax during rehabilitation training is expected to improve dynamic knee stability and support patients' safe return to sports.

In the ACL-injured group, the peak EMG values of the quadriceps muscles (VL, VM) were significantly lower than those of the healthy control group within 100 ms after landing, suggesting that the activation of the knee extensor muscles during the touchdown phase was reduced. This feature is referred to as the "quadriceps avoidance strategy", reflecting the fact that patients may actively reduce extensor output to avoid knee instability, which affects joint braking and postural control[38]. In contrast, the hamstrings showed a tendency to increase activation, especially ST, which peaked significantly within 100 ms before touchdown, and BF, which tended to increase both before and after touchdown. As a co-stabilizing muscle of the ACL, popliteus mainly limits anterior translation and internal rotation by generating tibial posterior moment, and its enhanced recruitment contributes to knee stability and compensates for the lack of ACL function. This feature fits with the electromechanical delay time of the hamstrings, which ensures the provision of an effective response during the peak phase of shear[32]. Gmax, although showing a reduced peak after landing in the injury group, has an earlier onset and longer duration, suggesting that it compensates through temporal regulation to maintain hip and pelvic stability. Hamstrings take on more loads when the function of Gmax is reduced, and their enhanced activation may also partly stem from the indirect compensation for gluteal insufficiency[39]. In addition, ACL injury may interfere with central drive, resulting in decreased efficiency of motor unit recruitment, further exacerbating Gmax underactivation[40]. Overall, the ACL-injured group showed an activation pattern of "extensor inhibition - flexor strengthening - gluteal sequential compensation", which reflects the self-regulation mechanism of the neuromuscular system under the condition of impaired joint stability. Clinically, quadriceps avoidance strategies are often associated with subjective instability and decreased functional jumping performance. However, enhanced activation of the hamstrings and Gmax may assume a new dynamic stabilizing function after ACL loss, helping patients maintain joint stability. Therefore, during rehabilitation, long-term quadriceps inhibition should be avoided, and strength and control training of the hamstrings and Gmax should be strengthened to compensate for the mechanical deficiencies caused by ACL loss and improve clinical functional outcomes. Although the increased activation of the hamstrings and Gmax in this study was primarily interpreted as a compensatory response to ACL injury, whether this phenomenon also indicates that these muscle groups have assumed a new stabilizing role after ACL loss warrants further investigation. Numerous studies have shown that the hamstrings and Gmax play an important role in knee stability[12,41]. Following ACL injury, they may not simply be a compensatory response but rather assume a new dynamic stabilizing function. The hamstrings can unload the ACL by generating posterior tibial shear forces, while the gluteal muscles unload the ACL by resisting knee valgus moments[42], a function that is particularly important after ACL injury. To further validate this, future studies should incorporate biomechanical data to investigate the role of the hamstrings and Gmax in knee stability after ACL loss.

This study had a relatively limited sample size (21 patients with ACL injuries and 21 healthy controls). Statistically, this small sample size may have reduced power, leading to some true effects not reaching significance. It may also have magnified the influence of individual differences on the mean, increasing the instability of the results. Physiologically, the small sample size may have masked differences in myoelectric patterns within specific populations. The subjects were primarily younger. Neuromuscular adaptations in younger individuals may manifest as faster preactivation and greater force output, which may underestimate the degree of quadriceps avoidance or hamstring compensation in landing strategies of older individuals. Therefore, the generalizability of the present results to other age groups is limited. Furthermore, all ACL-injured patients in this study had their dominant leg injured. The dominant leg typically possesses greater strength and more mature motor control, potentially demonstrating earlier preactivation and longer sustained activation to maintain task stability. This characteristic may have magnified the observed compensatory trends in the hamstrings and Gmax; conversely, the magnitude and strategy of compensation may differ in the non-dominant leg. Therefore, the omission of the non-dominant leg limits our inferences about the relationship between limb dominance and EMG timing and intensity. Additionally, because this study was primarily exploratory in nature, p-values were not adjusted for multiple comparisons. The absence of p-value correction should be considered when interpreting the results. Future studies with larger and more diverse samples are warranted to validate these findings and, where appropriate, to adopt false discovery rate (FDR)-based approaches such as the Benjamini–Hochberg procedure to enhance statistical rigor, particularly in confirmatory research.

Conclusion

In a single-leg jump landing task, patients with ACL injuries of an average disease duration of 18 months generally had a prolonged muscle activation duration, reflecting compensatory mechanisms to maintain kinematic stability, despite a preactivation sequence consistent with that of healthy individuals. Preactivation of the biceps femoris, semitendinosus, and gluteus maximus muscles in the affected limb occurred with delayed hamstring peaks, potentially contributing to knee stability by modulating tibial motion and reducing excessive anterior translation and internal rotation.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Received: 10 May 2025; Accepted: 18 November 2025
Published online: 28 November 2025

References

1. Griffin, L. Y. et al. Understanding and preventing noncontact anterior cruciate ligament injuries: a review of the Hunt Valley II meeting, January 2005. *Am. J. Sports Med.* **34**, 1512 (2006).
2. Nordenvall, R. et al. A population-based nationwide study of cruciate ligament injury in Sweden, 2001–2009: incidence, treatment, and sex differences. *Am. J. Sports Med.* **40**, 1808 (2012).
3. Ao, Y., Yu, C. & Tian, D. Anterior cruciate ligament injury in female athlete. *Chin. J. Sports Med.* **19**, 387 (2000).
4. Baker, L. A. et al. Multivariate genome-wide association analysis identifies novel and relevant variants associated with anterior cruciate ligament rupture risk in the dog model. *BMC GENET.* **19**, 39 (2018).
5. Wright, R. W., Magnussen, R. A., Dunn, W. R. & Spindler, K. P. Ipsilateral graft and contralateral ACL rupture at five years or more following ACL reconstruction: a systematic review. *J. BONE JOINT SURG. AM.* **93**, 1159 (2011).
6. Grassi, A. et al. More than a 2-Fold risk of contralateral anterior cruciate ligament injuries compared with ipsilateral graft failure 10 years after primary reconstruction. *Am. J. Sports Med.* **48**, 310 (2020).
7. Olsen, O. E., Myklebust, G., Engebretsen, L., Holme, I. & Bahr, R. Exercises to prevent lower limb injuries in youth sports: cluster randomised controlled trial. *BMJ* **330**, 449 (2005).
8. Myklebust, G. et al. Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. *CLIN. J. SPORT MED.* **13**, 71 (2003).
9. Theisen, D., Rada, I., Brau, A., Gette, P. & Seil, R. Muscle Activity Onset Prior to Landing in Patients after Anterior Cruciate Ligament Injury: A Systematic Review and Meta-Analysis. PLOS ONE 11 e155277 (2016).
10. Zebis, M. K. et al. Effects of evidence-based prevention training on neuromuscular and Biomechanical risk factors for ACL injury in adolescent female athletes: a randomised controlled trial. *Br. J. Sports Med.* **50**, 552 (2016).
11. Wu, X. et al. Surface Electromyography and Gait Features in Patients after Anterior Cruciate Ligament Reconstruction. ORTHOP SURG (2024).
12. Rinaldi, V. G., Prill, R., Jahnke, S., Zaffagnini, S. & Becker, R. The influence of gluteal muscle strength deficits on dynamic knee valgus: a scoping review. *J. Exp. Orthop.* **9**, 81 (2022).
13. Ito, N., Capin, J. J., Khandha, A., Buchanan, T. S. & Snyder-Mackler, L. Identifying gait pathology after ACL reconstruction using Temporal characteristics of kinetics and electromyography. *Med. Sci. Sports Exerc.* **54**, 923 (2022).
14. Georgoulis, J. D. et al. Neuromuscular activity of the lower-extremities during running, landing and changing-of-direction movements in individuals with anterior cruciate ligament reconstruction: a review of electromyographic studies. *J. Exp. Orthop.* **10**, 43 (2023).
15. Trulsson, A., Miller, M., Gummesson, C. & Garwicz, M. Associations between altered movement patterns during single-leg squat and muscle activity at weight-transfer initiation in individuals with anterior cruciate ligament injury. BMJ Open Sport Exerc Med **2** e131 (2016).
16. Cohen, J. *Statistical Power Analysis for the Behavioral Sciences* 2nd edn (Statistical Power Analysis for the Behavioral Sciences, 1988).
17. Kapreli, E. et al. Anterior cruciate ligament deficiency causes brain plasticity: a functional MRI study. *Am. J. Sports Med.* **37**, 2419 (2009).
18. Swanik, C. B., Lephart, S. M., Giraldo, J. L., Demont, R. G. & Fu, F. H. Reactive muscle firing of anterior cruciate ligament-injured females during functional activities. *J. Athl Train.* **34**, 121 (1999).
19. Stegeman, D. & Hermens, H. Standards for surface electromyography: The European project Surface EMG for non-invasive assessment of muscles (SENIAM). Enschede: Roessingh Research and Development **10**, 8 (2007).
20. Daanen, H. A., Mazure, M., Holewijn, M. & Van der Velde, E. A. Reproducibility of the mean power frequency of the surface electromyogram. *Eur. J. Appl. Physiol. Occup. Physiol.* **61**, 274 (1990).
21. DeLuca, P. A., Davis, R. R., Ounpuu, S., Rose, S. & Sirkin, R. Alterations in surgical decision making in patients with cerebral palsy based on three-dimensional gait analysis. *J. Pediatr. Orthop.* **17**, 608 (1997).
22. Nedergaard, N. J., Dalbø, S., Petersen, S. V., Zebis, M. K. & Bencke, J. Biomechanical and neuromuscular comparison of single- and multi-planar jump tests and a side-cutting maneuver: implications for ACL injury risk assessment. *KNEE* **27**, 324 (2020).
23. McCurdy, K., Walker, J. & Yuen, D. Gluteus Maximus and hamstring activation during selected Weight-Bearing resistance exercises. *J. STRENGTH. COND RES.* **32**, 594 (2018).
24. Kang, S. Y., Jeon, H. S., Kwon, O., Cynn, H. S. & Choi, B. Activation of the gluteus Maximus and hamstring muscles during prone hip extension with knee flexion in three hip abduction positions. *Man. Ther.* **18**, 303 (2013).
25. Steele, J. R. & Brown, J. M. Effects of chronic anterior cruciate ligament deficiency on muscle activation patterns during an abrupt deceleration task. *Clin. Biomech. (Bristol Avon).* **14**, 247 (1999).
26. Cowling, E. J. & Steele, J. R. Is lower limb muscle synchrony during landing affected by gender? Implications for variations in ACL injury rates. *J. Electromyogr. Kinesiol.* **11**, 263 (2001).
27. Bryant, A. L., Newton, R. U. & Steele, J. Successful feed-forward strategies following ACL injury and reconstruction. *J. Electromyogr. Kinesiol.* **19**, 988 (2009).
28. Smeets, A. et al. Single-Joint and Whole-Body movement changes in anterior cruciate ligament athletes returning to sport. *Med. Sci. Sports Exerc.* **52**, 1658 (2020).
29. Yoshii, R. et al. Abnormality in re-programing of preparatory muscle activity for landing following unpredictable events in patients with anterior cruciate ligament injury. *KNEE* **49**, 8 (2024).
30. Alvarez-Diaz, P. et al. Effects of anterior cruciate ligament injury on neuromuscular tensiomyographic characteristics of the lower extremity in competitive male soccer players. *Knee Surg. Sports Traumatol. Arthrosc.* **24**, 2264 (2016).
31. Lass, P. et al. Muscle coordination following rupture of the anterior cruciate ligament. Electromyographic studies of 14 patients. *Acta Orthop. Scand.* **62**, 9 (1991).
32. Knoll, Z., Kiss, R. M. & Kocsis, L. Gait adaptation in ACL deficient patients before and after anterior cruciate ligament reconstruction surgery. *J. Electromyogr. Kinesiol.* **14**, 287 (2004).
33. Zhou, S., Lawson, D. L., Morrison, W. E. & Fairweather, I. Electromechanical delay of knee extensors: the normal range and the effects of age and gender. *OAI* **28**, 3 (1995).
34. Blackburn, J. T., Bell, D. R., Norcross, M. F., Hudson, J. D. & Engstrom, L. A. Comparison of hamstring neuromechanical properties between healthy males and females and the influence of musculotendinous stiffness. J Electromyogr Kinesiol 19 e362 (2009).
35. Kim, J. W. et al. Age-sex differences in the hip abductor muscle properties. *GERIATR. GERONTOL. INT.* **11**, 333 (2011).
36. Koga, H., Nakamae, A., Shima, Y., Bahr, R. & Krosshaug, T. Hip and ankle kinematics in noncontact anterior cruciate ligament injury situations: video analysis using Model-Based image matching. *Am. J. Sports Med.* **46**, 333 (2018).

37. Krosshaug, T., Slauterbeck, J. R., Engebretsen, L. & Bahr, R. Biomechanical analysis of anterior cruciate ligament injury mechanisms: three-dimensional motion reconstruction from video sequences. *Scand. J. Med. Sci. Sports*. **17**, 508 (2007).
38. Wexler, G., Hurwitz, D. E., Bush-Joseph, C. A., Andriacchi, T. P. & Bach, B. J. Functional gait adaptations in patients with anterior cruciate ligament deficiency over time. *Clin. Orthop. Relat. Res.* **348**, 166 (1998).
39. Matsunaga, N. et al. Muscle fatigue in the gluteus Maximus changes muscle synergies during single-leg landing. *J. Bodyw. Mov. Ther.* **27**, 493 (2021).
40. Trulsson, A., Miller, M., Hansson, G., Gummesson, C. & Garwicz, M. Altered movement patterns and muscular activity during single and double leg squats in individuals with anterior cruciate ligament injury. *BMC Musculoskelet. Disord.* **16**, 28 (2015).
41. Maniar, N., Schache, A. G., Sritharan, P. & Opar, D. A. Non-knee-spanning muscles contribute to tibiofemoral shear as well as valgus and rotational joint reaction moments during unanticipated sidestep cutting. *Sci. Rep.* **8**, 2501 (2018).
42. Maniar, N., Cole, M. H., Bryant, A. L. & Opar, D. A. Muscle force contributions to anterior cruciate ligament loading. *SPORTS MED.* **52**, 1737 (2022).

Acknowledgements
The authors express gratitude to all participants.

Author contributions
J.X. and M.C. designed the research. J.X. and J.R. collected, analyzed the data, and drafted the manuscript. J.X. and XB. L. revised the manuscript. All authors contributed to the article and approved the submitted version.

Declarations

Competing interests
The authors declare no competing interests.

Additional information
Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-025-29516-y.

Correspondence and requests for materials should be addressed to X.L.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.