



Original research

Muscle atrophy contributes to quadriceps weakness after anterior cruciate ligament reconstruction



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ARTICLE INFO

Article history:

Received 18 January 2014

Received in revised form

11 December 2014

Accepted 17 December 2014

Available online 13 January 2015

Keywords:

Anterior cruciate ligament

Muscle

Strength

MRI

Central activation ratio

ABSTRACT

Objectives: Quadriceps weakness persists after anterior cruciate ligament reconstruction. Muscle atrophy and activation failure may contribute. This study examined the roles of atrophy and activation failure in quadriceps weakness after anterior cruciate ligament reconstruction.

Design: Case series.

Methods: Twenty patients six months post-anterior cruciate ligament reconstruction participated. Atrophy was determined as peak quadriceps cross sectional area from magnetic resonance images. Quadriceps activation was quantified via the central activation ratio, while muscle strength was measured isometrically. All testing was performed bilaterally. Hierarchical linear regression and one-way ANOVAs were performed to examine the relation of muscle strength with activation and atrophy.

Results: Cross sectional area ($R^2 = 0.307$; $p = 0.011$) explained more of the variance in quadriceps strength than central activation ratio ($R^2 < 0.001$; $p = 0.987$). Strength and cross sectional area were lower in the injured (strength: 2.03 ± 0.51 N m/kg; cross sectional area: 68.81 ± 17.80 cm²) versus uninjured limb (strength: 2.89 ± 0.81 N m/kg; cross sectional area: 81.10 ± 21.58 cm²; $p < 0.001$). There were no side-to-side differences in central activation ratio; however, quadriceps activation failure was present bilaterally (injured: 0.87 ± 0.12 ; uninjured: 0.85 ± 0.14 ; $p = 0.571$).

Conclusions: Quadriceps cross sectional area was strongly related to muscle strength six months after anterior cruciate ligament reconstruction and substantial injured versus uninjured limb deficits were demonstrated for strength and cross sectional area. Patients may benefit from exercises aimed at improving quadriceps cross sectional area post-operatively.

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1. Introduction

Quadriceps weakness is nearly ubiquitous following anterior cruciate ligament (ACL) injury and reconstruction (ACLR).¹ Strength deficits upwards of 30% in the reconstructed compared to the contralateral limb have been reported six months post-operatively,² a time when patients often return to full activity. The presence of quadriceps weakness may be hazardous to the patient. The quadriceps is important to lower limb control during dynamic activity and quadriceps weakness could alter movement strategies potentiating re-injury.³ To optimally prepare patients to return to full activity, complete quadriceps function must be restored. However, before

quadriceps strength deficits can be effectively countered, a deeper understanding of why quadriceps weakness persists throughout rehabilitation is needed.

Quadriceps disuse atrophy occurs following knee joint immobilization and may contribute to quadriceps weakness after ACL injury and reconstruction.⁴ Konishi and colleagues⁵ previously reported an approximately 7% deficit in total quadriceps volume in the reconstructed versus contralateral limb in patients 6–12 months following ACL reconstruction. Deficits of 3% in volume⁶ and cross sectional area⁷ (CSA) remained 12–18 months post-operatively. Similar magnitudes of quadriceps atrophy were reported by Lorentzon et al.⁸ in people with ACL deficiency, though no relation between atrophy and strength was identified. The authors concluded that muscle atrophy alone did not cause quadriceps weakness suggesting, instead, that incomplete volitional muscle activation may contribute.⁸

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Quadriceps activation failure (QAF) is a common consequence of ACL injury and reconstruction. QAF is often associated with joint damage, effusion, and pain.⁴ These factors alter the afferent signal sent to the central nervous system, which leads to an inhibitory signal transmitted to the quadriceps α -motoneuron pool and a decrease in voluntary muscle activity.⁴ Previous reports of QAF following ACLr suggest deficits upwards of 15% present two years post-operatively.¹ In patients undergoing total knee arthroplasty, a population with similar magnitude quadriceps dysfunction to those following ACL injury,⁹ QAF accounted for nearly twice the quadriceps strength deficit as muscle atrophy in the acute post-operative period.⁹ Elucidating how quadriceps muscle atrophy and QAF contribute to lingering weakness when patients return to full activity seems imperative. Thus, the purpose of this study was to determine if quadriceps atrophy and QAF contribute to persistent knee extension strength deficits in patients when they were cleared to return to full activity after ACL reconstruction. We hypothesized that persistent quadriceps weakness would result from a combination of QAF (measured by central activation ratio [CAR]) and muscle atrophy (measured through cross sectional area [CSA]) and that QAF would more strongly predict quadriceps weakness than would muscle atrophy. We further hypothesized that greater quadriceps weakness, QAF, and CSA would be present in the injured compared with the uninjured limb.

2. Methods

Twenty-two patients were recruited for participation; one was excluded after secondary screening revealed she did not fulfill all of the inclusion criteria. Another individual reported for magnetic resonance imaging (MRI) testing but failed to report for CAR assessment. He could not be reached for follow-up and was excluded from analysis, leaving 20 patients (7 males, 13 females; age: 20.65 ± 5.17 years; height: 1.72 ± 0.08 m; mass: 74.47 ± 14.49 kg) who underwent patellar tendon autograft ACL reconstruction. Patients received physician clearance for return to full activity prior to enrollment.

Patients reported for testing on two occasions (212.89 ± 31.62 days post-ACL reconstruction), with quadriceps muscle atrophy, in the form of muscle CSA measured at one session and strength and CAR measured at the other (12.89 ± 16.63 days between testing sessions). All measures were assessed bilaterally.

Potential patients were excluded if they had: (1) a history of lower extremity surgery other than their recent ACLr; (2) current pain in either knee; (3) a partial or complete meniscectomy with their ACLr; (4) other ligamentous damage concurrent with their ACL injury; or (5) a known heart condition. Pregnant females were also excluded. This study was approved by the medical school institutional review board.

Patients rated their knee symptoms and function using the 2000 International Knee Documentation Committee (IKDC) subjective form. IKDC scores can range from 0 to 100, with lower scores indicating worse symptoms and functional impairments. The Tegner scale was utilized to determine physical activity levels. The IKDC and Tegner scales were completed when patients reported for strength and QAF testing.

To assess quadriceps muscle atrophy, patients lay supine in a MRI scanner (Philips Achieva 3T Quasar Dual, Philips Electronics, Andover, MA, USA) for simultaneous, bilateral thigh scans. The proton density-weighted images had the following parameters: repetition time 200–3000 ms, echo time 35 ms, slice thickness 6 mm, gap between slices 6 mm, matrix 364×180 , and field of view $480 \text{ mm} \times 281 \text{ mm}$.

Peak CSA for each of the quadriceps muscles, and total quadriceps peak CSA, was evaluated. The contours of each muscle

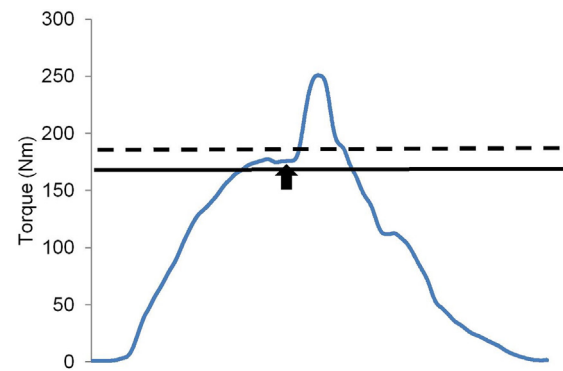


Fig. 1. Screenshot from strength and QAF testing. The blue line represents the patient's real-time torque output. The solid black line corresponds to the patient's peak value from the MVIC trials and also serves as a threshold for QAF testing. Real-time torque output must cross threshold for the electrical stimulus to be delivered. The dotted line represents the patient's target value, which was set 10% above maximal strength. The black arrow corresponds to delivery of the electrical stimulus. QAF = central activation failure. MVIC = maximal voluntary isometric contraction. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

were traced in every axial image in which the muscle appeared using ImageJ software (version 142q, National Institutes of Health, Bethesda, MD, USA) and an Intuos4 pen tablet (Wacom Technology Corporation, Vancouver, WA, USA). The sum of each muscle's CSA yielded the total CSA for each slice. The slice with the greatest total CSA was used for statistical analysis.⁹ All atrophy measurements were performed by a single investigator with high intrarater reliability (intraclass correlation coefficient of 0.988).

Joint effusion was measured while patients lay supine and the superior pole of the patella was located by palpation. A mark was placed 1 cm proximal to the superior pole and the circumference of the knee at this location was obtained with a cloth tape measure.¹⁰

Quadriceps strength was assessed using knee extension maximal voluntary isometric contractions (MVICs) while patients were seated on an isokinetic dynamometer (Biodex System 3, Biodex Medical Systems, Shirley, NY, USA) with the hip flexed to 85° and the knee flexed to 90° . For MVIC testing, patients were instructed to kick out as hard as they could while watching a computer monitor running a custom-written Labview (version 8.5, National Instruments, Austin, TX, USA) program that displayed their real-time torque output. After completion of the first MVIC, the program displayed a solid line reflecting the patient's peak torque value from the initial trial and a dashed line set 10% above the peak torque recorded during the initial trial (Fig. 1). For subsequent trials, patients were encouraged to reach the target torque value (dashed line). If patients increased their torque during any ensuing trial, the height of the solid and dashed lines was adjusted accordingly. A minimum of three knee extension MVICs were performed, with at least 2 min of rest in between repetitions, until no improvements in torque were observed by an investigator. Once peak torque was reached, the highest torque value from all recorded repetitions was noted and used as a threshold for QAF assessment.

For QAF testing, self-adhesive, stimulating electrodes (Dura-Stick II [5 cm \times 9 cm] Chattanooga Group, Hixson, TN, USA) were applied proximally over the rectus femoris and distally over the vastus medialis. At the beginning of QAF testing, the peak torque value from the MVIC trials was input into the custom-written program. The program utilized this threshold (peak torque) value to determine whether or not to trigger the electrical stimulator (S88 and SIU8T, GRASS Technologies, West Warwick, RI, USA) to deliver a stimulus (100 ms-long train, 600 μ s pulse duration, 100 pps deliver rate, 130 V maximal voltage). Similar to MVIC testing, patients were instructed to generate enough torque to reach the dashed target

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