Strengthening and Neuromuscular Reeducation of the Gluteus Maximus in a Triathlete With Exercise-Associated Cramping of the Hamstrings

Exercise-associated muscle cramping (EAMC) is a common problem amongst triathletes and marathon runners. Triathletes experience a lifetime prevalence of muscle cramping of 67%, and marathon runners one of 30% to 50%. The literature reports that muscles crossing 2 joints (ie, hamstrings) are more prone to EAMC than muscles that cross 1 joint. Despite attempts at management, this condition often persists and athletes have difficulty returning to competition without recurrence.

Researchers discuss 2 theories concerning the etiology of EAMC: (1) local muscle overload and (2) electrolyte deficit. As such, interventions for EAMC typically consist of stretching/strengthening of the involved muscle and/or supplements to restore electrolyte imbalances.

**CASE DESCRIPTION:** The patient was a 42-year-old male triathlete with a primary complaint of recurrent cramping of his right hamstring muscle, which prevented him from completing races at his desired pace. Strength testing revealed gluteus maximus muscle weakness bilaterally. Electromyographic (EMG) analysis (surface electrodes, 1560 Hz) revealed that the right hamstrings were being activated excessively during terminal swing and the first half of the stance phase (48.1% maximum voluntary isometric contraction [MVIC]).

**STUDY DESIGN:** Case report.

**OBJECTIVE:** To highlight the effects of an intervention program consisting of strengthening and neuromuscular reeducation of the gluteus maximus in an elite triathlete with exercise-associated muscle cramping (EAMC).

**BACKGROUND:** Researchers have described 2 theories concerning the etiology of EAMC: (1) muscle fatigue and (2) electrolyte deficit. As such, interventions for EAMC typically consist of stretching/strengthening of the involved muscle and/or supplements to restore electrolyte imbalances.

**CASE DESCRIPTION:** The patient was a 42-year-old male triathlete with a primary complaint of recurrent cramping of his right hamstring muscle, which prevented him from completing races at his desired pace. Strength testing revealed gluteus maximus muscle weakness bilaterally. Electromyographic (EMG) analysis (surface electrodes, 1560 Hz) revealed that the right hamstrings were being activated excessively during terminal swing and the first half of the stance phase (48.1% maximum voluntary isometric contraction [MVIC]).

**OUTCOMES:** Following the intervention, the patient was able to complete 3 triathlons without hamstring cramping. Strength testing revealed that the right hip extension strength improved from 35.6 to 54.7 kg, and activation of the hamstrings during terminal swing and the first half of the stance phase decreased to 36.4% of MVIC.

**DISCUSSION:** A program of gluteus maximus strengthening and neuromuscular training eliminated EAMC of the hamstrings in this patient. Given that the hamstrings and gluteus maximus work as agonists to decelerate the thigh during terminal swing phase and control hip flexion during loading response of running, we postulate that strengthening the gluteus maximus decreased the relative effort required by the hamstrings, thus reducing EAMC. The results of the EMG evaluation that was performed as part of this case report provides support for this hypothesis.

**LEVEL OF EVIDENCE:** Therapy, level 4.

**KEY WORDS:** hip, lower extremity, muscle cramping, running

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...ent activity of type Ia and type II muscle spindles and a decrease in afferent activity of type Ib/Golgi tendon organs. This increase in excitatory responses (type Ia, II) and decrease in inhibitory responses (type Ib/Golgi tendon organs) has been hypothesized to result in sustained alpha motor neuron activity indicative of muscle cramping.2,22,26-30

Evidence suggests that EAMC also can be caused by an electrolyte deficit. Electrolyte deficit is caused by excessive sweating, leading to a loss of sodium and/or chloride, as well as a decrease in plasma volume. The result is a “whole body exchangeable sodium deficit” that can occur when 20% to 30% total body sodium is lost.2 This theory suggests that as fluid leaves the interstitial space to restore plasma volume, neuromuscular junctions are exposed to heightened levels of excitatory elements, which ultimately leads to increased initiation of action potentials.2 Sjøgaard et al21 have reported that in such a scenario, increased concentrations of sodium in interstitial spaces could increase action potentials by decreasing the depolarization threshold. Cramping of this origin begins with light fasciculations and is followed by more severe muscle spasms.

The loss of potassium, calcium, and magnesium also is thought to contribute to EAMC. However, studies by Sulzer23 and Maughan22 reported that prerace and postrace serum concentrations of these minerals did not differ among athletes with or without EAMC. Potassium, magnesium, and calcium are lost to a lesser extent with excessive sweating and, as such, have not been implicated as prominently in the electrolyte deficit theory of EAMC as sodium and chloride.2

One possible reason for limited long-term success with previous interventions for EAMC may be related to the failure to identify the correct etiology of the problem. Theoretically, EAMC resulting from local muscle fatigue will not respond to attempts to restore electrolyte imbalances. Conversely, EAMC resulting from an electrolyte imbalance will not respond to interventions directed to the local muscles such as soft tissue mobilization, stretching, and strengthening.2,28-30

This case report describes the management of a triathlete with recurrent EAMC of the hamstrings during the running phase of competition. We suspected that EAMC was the result of local muscle fatigue, as numerous attempts to modify fluid/sodium intake were not effective in alleviating symptoms. During running, one role of the hamstrings is to eccentrically decelerate hip flexion and knee extension during terminal swing (ie, just prior to initial contact). Additionally, the hamstrings help control hip flexion during loading response.15,23 The functions of the hamstrings during running are assisted by the gluteus maximus, which also acts to decelerate hip flexion during terminal swing and to control trunk and hip flexion during loading response. Given that the hamstrings and gluteus maximus are agonists,15,23 it is conceivable that weakness of the gluteus maximus could increase the relative effort of the hamstring muscles, leading to overuse, premature fatigue, and cramping.

The purpose of this case report was to highlight the effects of strengthening and neuromuscular reeducation of the gluteus maximus in treating recurrent cramping of the hamstrings. It was our clinical hypothesis that improving the gluteus maximus muscle performance would result in decreased hamstring activation in terminal swing and the first half of the stance phase of running, and thus decrease EAMC.

CASE DESCRIPTION

General Demographics

The patient was a 42-year-old male, with a height of 180.5 cm, body mass of 66.9 kg, and a body mass index of 20.6 kg/m². He was an elite-level triathlete who had competed in full- and half-ironman competitions for 12 years. At the time of his initial examination he stated that he was training 6 days a week.

History of Presenting Condition

The patient described a history of hamstring cramping pain that began during the Ironman Hawaii Triathlon in 2003. He stated that for 4 years his symptoms had not resolved and that he still experienced intermittent cramping that alternated between the right and left hamstrings. Over the past year, he reported right hamstring cramping in every race in which he participated.

The patient was seen by a physical therapist 4 years ago and received treatment consisting of hamstring stretching and cross-friction soft tissue mobilization. He stated that his onset, frequency, and intensity of cramping pain did not change after his participation in physical therapy. Over the past 4 years he made several attempts to modify fluid/sodium intake prior to races, but this had no effect on his symptoms. At the time he presented to us for treatment, he was performing stretches for the hip flexors, quadriceps, and hamstrings as previously advised by a physical therapist.

Presenting Complaints

The patient complained of right hamstring cramping that would occur during the running portion of his triathlons. He described a precipitating sensation of increasing tightness that would lead to painful cramping. He reported that the cramping sensation would worsen during downhill running and with increasing running speed. To alleviate cramping during a race, he would stop running and stretch his hamstrings until the cramping subsided. He would then continue running at a slower pace using a shuffling walk/run pattern. The patient stated his goals were to eliminate his hamstring cramping and to complete a half-ironman triathlon in under 4 hours 30 minutes.

Tests and Measures

Medical Screening Medical history screening was negative for any red flags, including bowel/bladder changes, discoordination of the lower extremities, and numbness or tingling in the saddle area,
hands, or feet. Medical screening also was negative for disease suggestive of systemic or nonmusculoskeletal pathology (ie, history of cancer, unexplained changes in weight, recent fever, or trauma). Past medical history was negative for previous surgeries, hypertension, and diabetes mellitus.

**Differential Diagnosis Screening** A lower extremity neurological screen was negative. Active, passive, and accessory motion of the lumbar spine, hip, and sacroiliac joint were assessed. These tests were conducted to rule out dysfunction of the following structures as the cause of the patient’s symptoms: (1) lumbar spine radiculopathy, (2) lower limb nerve mobility deficit, (3) lumbar facet dysfunction, (4) sacroiliac joint dysfunction, and (5) hip joint dysfunction.

**Posterior Thigh Examination** Hamstring palpation examination was performed to confirm the location of symptoms. Tenderness was noted with palpation of the middle portion of the long head of the biceps femoris and semitendinosus muscles on the right.

Muscle flexibility clinical tests were performed to determine the length of the hamstrings muscles bilaterally. Gonio metric measurements were obtained by the same clinician preintervention and postintervention. Hamstring length was measured with the patient supine and the hip flexed to 90°. The knee was then extended passively to the limit of tissue resistance and the knee flexion angle relative to the neutral position (ie, full knee extension) was measured. The average of 2 measurements for the left and right sides were 25° and 29°, respectively. These values were indicative of moderate muscle length restriction based on the normative threshold of 20° proposed by Magee.

Muscle strength tests for hip extension and knee flexion were performed using a Microfet2 handheld dynamometer (Hoggan Health Industries, Inc, Draper, UT). Assessing strength using a handheld dynamometer has been shown to be valid, with good to excellent intratester reliability.

Hip extension strength was assessed in the modified test position, as described by Kendall et al with the patient prone and the knee flexed to 70°. Resistance was provided at the distal thigh proximal to the popliteal fossa. Dynamometer testing revealed readings of 35.5 and 35.6 kg for the left and right sides, respectively.

Knee flexion strength was assessed in test position, as described by Kendall et al, with the patient prone and the knee flexed to 70°. Resistance was provided at the distal calf. Dynamometer testing revealed readings of 26.1 and 27.2 kg for the left and right sides, respectively. Right knee flexion strength testing resulted in hamstring cramping. The left side did not cramp during strength testing.

**Dynamic Assessment** Dynamic function was assessed during gait, lunge walking, and a step-down task. The purpose of the dynamic functional testing was to identify potential movement patterns that might have been contributing to the patient’s condition. Each activity represented an increasing amount of difficulty with respect to the control of lower limb alignment.

Observational gait analysis was performed as described by Perry. The patient was instructed to walk at a self-selected pace along a 10-m walkway. Notable sagittal plane abnormalities included inadequate knee extension in midstance and terminal swing bilaterally. In midstance, normal motion consists of the knee moving from 15° of flexion to full knee extension. In terminal swing, normal motion consists of the knee moving from 30° of flexion to full extension just prior to initial contact. The patient demonstrated approximately 20° of knee flexion in midstance and 10° of knee flexion at the end of terminal swing. Frontal or transverse plane gait deviations, such as hip adduction or internal rotation, were not observed. The patient did not complain of any symptoms during gait.

The patient was instructed in lunge walking. He performed consecutive lunges along a 10-m walkway. During this activity, the patient exhibited excessive hip adduction and internal rotation on the right (resulting in the knee joint center to deviate medially with respect to the foot), and a right trunk lean at maximum knee flexion of the lead lower extremity. These deviations were observed to a lesser extent on the left side.

Next, the patient was examined during a single-limb step-down test. He was instructed to lower slowly from a 20.32-cm-high (8-in) step and return to the starting position over the course of 6 seconds (3 seconds to lower his body and 3 seconds to raise his body). This task was completed to the beat of a metronome. From the frontal view, the patient demonstrated excessive internal hip rotation and adduction, and a right trunk lean when tested for the right side (Figure 1A). From the lateral view, he demonstrated diminished hip flexion and increased ankle dorsiflexion, and forward displacement of the knee relative to the toes on the right side.

**Assessment** Information gathered during history intake and physical exam led us to speculate that our patient’s EAMC was consistent with the skeletal muscle fatigue theory. Our clinical hypothesis was based primarily on the finding of diminished hip extensor muscle performance. Although manual muscle testing of hip extension cannot differentiate the contribution of the gluteus maximus and hamstrings, the patient demonstrated excessive hip internal rotation and adduction during dynamic testing, suggesting that gluteus maximus muscle performance was impaired. Given that the hamstrings and gluteus maximus are agonists during running, we theorized that weakness and/or impaired neuromuscular control of the gluteus maximus was increasing the relative effort required by the hamstring muscles during terminal stance and the first half of the stance phase, leading to overuse, premature fatigue, and cramping.
At this point, a decision was made to refer the patient to the Musculoskeletal Biomechanical Research Laboratory at the University of Southern California for electromyographic (EMG) evaluation to test this hypothesis. The purpose of this testing was to document the patient’s right hamstring activation during running to provide objective data for post-treatment comparisons. We theorized that if the gluteus maximus strength increased, hamstring recruitment during running should decrease.

**Biomechanical Evaluation**

EMG signals of the right hamstring muscles were recorded at 1560 Hz, using double-differential, preamplified, bipolar, grounded surface electrodes (Motion Control, Salt Lake City, UT). Each electrode consisted of 2 circular stainless contacts (12-mm diameter) separated by a distance of 17 mm. Prior to electrode placement, the skin was shaved, abraded with coarse gauze to reduce skin impedance, and cleaned with isopropyl alcohol. Consistent with a previous publication, electrodes were placed over the semimembranosus and biceps femoris muscle bellies midway between the ischial tuberosity and the medial/lateral epicondyles of the femur.

Data were obtained while the subject ran at a self-selected speed of 7 m/s along a 15-m walkway. A force plate (AMTI Corp, Newton, MA) was used to determine the timing of foot strike. Ground reaction force data were collected at 1560 Hz and synchronized with the EMG signals. Data obtained from running 3 trials were averaged for analysis.

EMG signals were band pass filtered (20-500 Hz), and a 60-Hz notch filter was applied. Data were full-wave rectified and a moving-average smoothing algorithm (75-millisecond window) was used to generate a linear envelope. EMG processing and smoothing was performed using EMG Analysis software (Motion Lab Systems, Baton Rouge, LA).

All EMG signals were normalized to the maximum EMG signal recorded during a maximum voluntary isometric contraction (MVIC). Patient positioning during the MVIC was the same as described above to assess knee flexion strength. The EMG variable of interest was the average hamstring EMG during the period of terminal swing (400 milliseconds prior to foot contact) through the first 50% of the stance phase. Data from the biceps femoris and semimembranosus were averaged over this period. Results revealed that the hamstrings were being activated at 48.1% MVIC, which is considered high for running.

**Intervention**

**Foundations for Treatment** The patient attended physical therapy once a month for 8 months. He was educated regarding his condition and the intended treatment approach. In addition, realistic goal setting was discussed.

The patient’s physical therapy program focused on strengthening and neuromuscular reeducation of the gluteus maximus, with exercises being progressed over 3 phases. The first phase consisted of non–weight-bearing exercises to emphasize isolated muscle recruitment. The second phase of the program consisted of weight-bearing exercises, and the third phase consisted of dynamic and ballistic training (ie, plyometrics). For each phase, exercises were performed on the right and left lower extremity.

Variable training parameters were used during each phase of the exercise program. In the first phase, exercises focused on muscle recruitment (3 sets of 8 to 15 repetitions were prescribed, with 1 to 2 minutes of rest between each set). In the second phase, exercises focused on muscle hypertrophy (3 to 5 sets of 4 to 8 repetitions were prescribed, with 2 to 3 minutes of rest between each set). In the third phase, exercises focused on muscle endurance (2 to 3 sets of 12 to 20 repetitions were prescribed, with 30 seconds to 3 minutes of rest between each set).

When the patient could complete the proposed exercises and repetitions in each phase, the program would progress to the next phase. The patient was given a home exercise program that he was instructed to perform once a day. The home exercise program paralleled the exercises given in the clinic and took approximately 20 minutes to complete.

During the course of his treatment, the patient continued with his regular triathlon training. In addition, the patient engaged in resistive weight training, and performed hip flexor stretches 2 times per week. He participated in races as scheduled.

**Phase 1: Isolated Muscle Recruitment (Weeks 0–4)** Prior to initiating the dynamic strengthening program of the gluteus maximus, the patient performed isolated contractions of this muscle with...
verbal and tactile cueing. Three exercises were prescribed during this phase.

The side-lying clam exercise was performed with the hip and knee in approximately 45° of flexion with the feet together (FIGURE 2A). The patient was instructed to raise his knee up and back, which was achieved through hip abduction and external rotation. The patient was told to keep his feet together and to not allow his trunk or pelvis to posteriorly rotate during the exercise.

Isometric gluteus maximus exercises were performed as described by Sahrmann.26 In the prone position, with 2 pillows under the abdomen and 1 knee flexed to 90° (FIGURE 2B), the patient performed an isometric gluteus maximus contraction. The patient rested his foot against a wall to minimize active knee flexion, in an attempt to reduce hamstring muscle recruitment. The patient progressed to active hip extension when he could initiate and maintain an isometric contraction for 10 seconds for 10 repetitions.

Lastly, a triplanar exercise was performed in quadruped, with the hip and knee in 90° of flexion. The patient demonstrated simultaneous hip abduction, external rotation, and extension without rotation of the lumbar spine or pelvis (FIGURE 2C).

Phase 2: Weight-Bearing Strengthening (Weeks 5-16) By week 4, the patient was progressed to weight-bearing exercises. These exercises consisted of dynamic side-squats, the star excursion exercise, forward step-downs, and forward/backward lunges. During all weight-bearing activities, the patient was encouraged to maintain neutral lower extremity alignment (NLEA). This involved positioning the lower extremity such that the anterior superior iliac spine and the knee remained positioned over the second toe.

While maintaining NLEA, the patient performed dynamic side-squats. The patient assumed a squat position of approximately 45° of hip and knee flexion (FIGURE 3A). Thera-Band positioned at the level of the mid thigh was used to resist bilateral hip abduction and external rotation during this exercise. The patient stepped to the right and left along a 10-m walkway by abducting and externally rotating the hips, with feet shoulder width apart for each step.

The next exercise performed in this phase was the star excursion exercise (FIGURE 3B). The patient was instructed to assume a partial single-limb squat position, then to reach as far as possible with the contralateral heel in 3 directions (forward, forward diagonal, and lateral) and to reach as far as possible with the
contralateral great toe in 2 directions (backward diagonal and backward). The depth of each squat on the stance limb was determined by the patient’s ability to maintain NLEA.

As the patient gained stability in single-limb stance for the star excursion exercise, the exercise was progressed to the forward step-down exercise (FIGURE 3C). For the step-down exercise, the patient started with a 5-cm (2-in) step. The patient was instructed to perform the exercise with NLEA during the descending and elevation portions of the movement. When the patient was able to demonstrate adequate hip control with the 5-cm step, the exercise was progressed in 5-cm increments up to a 20.32-cm (8-in) step.

Forward and backward lunges were performed with Thera-Band positioned at the mid-thigh level (FIGURE 3D). When performing the lunges, the patient was instructed to flex his knee to a depth of 75° and to not allow his knee to pass beyond his foot. The patient was encouraged to maintain a stable pelvis position during all lunging activities and was provided visual feedback through the use of a mirror.

Phase 3: Functional Training (Weeks 17-24) Once the patient completed the exercises outlined in phase 2, he was introduced to double-limb and single-limb plyometric activities. The patient was again educated on maintaining NLEA during the initiation and landing of all jumps and was provided visual feedback with use of a mirror. The exercises in this phase included double- and single-limb vertical and forward jumping, and an exercise for eccentric recruitment of the gluteus maximus during the swing phase of running.

The patient performed a double-limb vertical jump with maximal effort and landed in a deep squat, while maintaining NLEA (FIGURE 4A). He was then progressed to double-limb forward jumping along a 10-m walkway. Next, the patient performed a single-limb vertical jump with maximal effort and landed while maintaining NLEA (FIGURE 4B). This exercise also was progressed to single-limb forward jumping along a 10-m walkway.

For the last exercise in this phase, the patient was instructed to simulate the swing phase of running (FIGURE 4C). The hip was moved from 10° of extension to 40° of flexion, with the knee held in 20° of flexion. In an attempt to prevent overdominance of the hamstring muscles, tactile cueing was provided (via palpation) to facilitate eccentric contraction of the gluteus maximus. The speed of the swing limb was progressively increased over time through verbal cueing.

OUTCOMES

At the end of the intervention, the patient met his stated goal by completing 3 half-ironman triathlons without hamstring cramping. The patient reported that he performed a personal best for the bike portion of one of these courses, and equaled his best time for the bike and swim portion of a race that was set 8 years earlier. He was able to complete 1 of the half-ironman triathlon courses in 4 hours and 58 minutes; however, this was 28 minutes longer than his stated goal.

Tests of muscle strength, flexibility, and dynamic functional assessments were repeated using the same tester and equipment as described above. Postintervention dynamometer testing revealed that right hip extension strength increased from 35.6 to 54.7 kg and left hip extension strength increased from 35.5 to 46.8 kg. Right knee flexion strength changed from 27.2 to 25.5 kg. Left knee flexion strength

FIGURE 4. Phase 3 exercises: (A) double-limb vertical/forward jumps, (B) single-limb vertical/forward jumps, and (C) simulated swing phase exercise.
increased from 26.1 to 32.4 kg. With the patient in the 90/90 test position, right hamstring length increased from 29° to 17° short of full knee extension. The left hamstring length increased from 25° to 18° short of full knee extension (TABLE). Qualitative improvements in gait, lunge walking, and step-downs (FIGURE 1B) also were observed. More specifically, the patient demonstrated a neutral hip position during all tests. Posttreatment EMG analysis revealed that activation of the hamstrings during terminal swing and the first half of the stance phase of running decreased from 48.1% MVIC to 36.4% MVIC.

**DISCUSSION**

This case report describes the management of a triathlete with a chief complaint of hamstring cramping during competition, who responded well to a program that emphasized gluteus maximus muscle performance. Clinical and functional goals were achieved with an intervention approach that did not include traditional interventions for EAMC, such as electrolyte supplementation, soft tissue mobilization, stretching, or strengthening of the involved muscle.

Bergeron and Schwellnus both discuss the role of muscle fatigue in EAMC. The gluteus maximus muscle and hamstring muscles are agonists and share common functions during running. More specifically, these muscles decelerate the thigh during terminal swing and resist hip flexion during early stance. In this case report, we suspected that EAMC was the result of hamstring overuse, resulting in premature muscular fatigue and subsequent cramping. The muscle fatigue theory is supported by the fact that our patient only experienced cramping during the run portion of his triathlons (after completing the bike and swim events).

There is consensus in the literature that hamstring activation occurs in the late swing and early stance phases of running. In this case report, preintervention EMG recordings of the hamstrings during terminal swing and the first half of the stance phase of running revealed average levels of hamstring activity (48.1% MVIC), well above what is considered normal for running. Based on EMG recordings, Pinnington et al. reported the average hamstring activity during terminal swing and the first half of the stance phase of running to be 19% MVIC. This supported our belief that the hamstrings were being overused in our patient. In an attempt to resolve the issue of hamstring fatigue and overuse, impaired gluteus maximus muscle function was addressed. Consistent with our clinical hypothesis, gluteus maximus strength increased and hamstring activation decreased postintervention. This implies that the relative effort of the hamstrings was reduced following the intervention. However, it should be noted that postintervention hamstring activation (36.4% MVIC) was still higher than the normative levels reported by Pinnington et al.

The results of the current case report are consistent with the findings of Sherry and Best, who demonstrated that a rehabilitation program focusing on hip and trunk strengthening/stability was far superior for return to sport following acute hamstring strains than a traditional rehabilitation program consisting of stretching and strengthening of the hamstring muscles. Both our case report and Sherry and Best’s clinical trial addressed impairments of the pelvis/trunk musculature to resolve a hamstring injury. This suggests that successful interventions for hamstring injuries do not need to be focused on the muscle itself.

The literature reports that shortened muscles and muscles that cross 2 joints are more prone to cramping as a result of muscle fatigue as opposed to electrolyte imbalance. Upon evaluation, our patient had moderately limited hamstring length, which suggests that this muscle group was more susceptible to cramping. Interestingly, hamstring length increased, despite no increase in his minimal stretching routine. Therefore, it could be argued that the decrease in muscle cramping experienced by our patient could have been influenced by an inadvertent increase in hamstring length through active lengthening. Given the lack of conclusive evidence in the literature to support hamstring stretching as an effective long-term solution to EAMC, we feel that it is unlikely that the

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**TABLE**

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*Strength measurements were performed using a handheld dynamometer. Hamstring measurements were performed in the 90/90 position and reflect the degrees from full knee extension.*
observed increase in hamstring length was responsible for the favorable results in our patient. A positive outcome in our patient was achieved with an intervention that focused on strengthening and neuromuscular reeducation of the gluteus maximus. As it stands, it is not clear how much the positive outcome in this case report was related to improved neuromuscular control versus increased strength. If the patient had not presented with impaired strength, one could argue that improved neuromuscular control could have had a more singular effect on our patient. Although, the focus of our intervention was strengthening of the gluteus maximus, other muscles were likely strengthened as well (glutes medius, piriformis, trunk musculature, etc.). As such, care must be taken in attributing the effects of the intervention solely to improved gluteus maximus muscle performance.

CONCLUSION

This case report suggests that a rehabilitation program with a focus on increasing gluteus maximus muscle performance eliminated running-related EAMC in a triathlete. Improving strength and neuromuscular control of the gluteus maximus resulted in a decrease in hamstring activation during terminal swing and the first half of the stance phase of running. We propose that strength of the gluteus maximus muscle should be assessed when evaluating a patient with hamstring EAMC. Despite the outcomes presented in this case report, care must be taken in establishing cause and effect, based on a single patient.

REFERENCES