Diminished Foot and Ankle Muscle Volumes in Young Adults With Chronic Ankle Instability

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Background: Patients with chronic ankle instability (CAI) have demonstrated altered neuromuscular function and decreased muscle strength when compared with healthy counterparts without a history of ankle sprain. Up to this point, muscle volumes have not been analyzed in patients with CAI to determine whether deficits in muscle size are present following recurrent sprain.

Purpose: To analyze intrinsic and extrinsic foot and ankle muscle volumes and 4-way ankle strength in young adults with and without CAI.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: Five patients with CAI (mean age, 23.0 ± 4 years; 1 male, 4 females) and 5 healthy controls (mean age, 23.8 ± 4.5 years; 1 male, 4 females) volunteered for this study. Novel fast-acquisition magnetic resonance imaging (MRI) was used to scan from above the femoral condyles through the foot and ankle. The perimeter of each muscle was outlined on each axial slice and then the 2-dimensional area was multiplied by the slice thickness (5 mm) to calculate the muscle volume. Plantar flexion, dorsiflexion, inversion, and eversion isometric strength were measured using a handheld dynamometer. Patients with CAI were compared with healthy controls on all measures of muscle volume and strength. Extrinsic muscle volumes of patients with CAI were also compared with a normative database of healthy controls ($n = 24$) by calculating z scores for each muscle individually for each CAI subject.

Results: The CAI group had smaller total shank, superficial posterior compartment, soleus, adductor hallucis obliqus, and flexor hallucis brevis muscle volumes compared with healthy controls as indicated by group means and associated 90% CIs that did not overlap. Cohen *d* effect sizes for the significant group differences were all large and ranged from 1.46 to 3.52, with 90% CIs that did not cross zero. The CAI group had lower eversion, dorsiflexion, and 4-way composite ankle strength, all with group means and associated 90% CIs that did not overlap. No other significant differences were identified.

Conclusion: Patients with CAI demonstrate atrophy of intrinsic and extrinsic foot and ankle musculature accompanied by lower ankle strength.

Clinical Relevance: Clinicians should be aware of the muscle atrophy and strength deficits when prescribing rehabilitation for patients with lateral ankle sprain or CAI.

Keywords: muscle morphology; ankle sprain; intrinsic foot muscles; strength

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Ankle sprains are the most common musculoskeletal injury and are estimated to account for 15% of all sport-related injuries.[24] Following an initial ankle sprain, up to 40% of individuals develop chronic ankle instability (CAI).[31] CAI is characterized by recurrent sprains, "giving way," persistent symptoms, and diminished self-reported function.[23] CAI has been linked to an increased rate of posttraumatic osteoarthritis and diminished quality of life.[32,39] Many patients with CAI are unable to maintain their previous physical activity level, and young adults with CAI have been observed to take over 2000 less steps per day than their healthy peers.[27]

Decreased physical activity,[32] neuromuscular dysfunction,[8,9,14,15] and decreased joint range of motion[10,13,23] are not only common characteristics of CAI but also well documented as potential causes of muscle atrophy.[4] Clinical manifestations of muscle atrophy could include muscle weakness, altered movement patterns, and increased risk

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of injury, all of which have consistently been reported in patients with CAI.[1,22,45] Although muscle morphology has not previously been investigated in patients with CAI, atrophy of the foot and ankle musculature has been identified in individuals with posttraumatic ankle osteoarthritis[44] and after foot and ankle immobilization.[38]

Invertor[45] and evertor[2] muscle strength are important for ankle sprain prevention. McKeon et al[34] recently highlighted the important synergistic interaction between the extrinsic foot muscles as global ankle joint movers and intrinsic foot muscles as local stabilizers during normal lower extremity function. Unfortunately, the complexity of the foot and ankle's numerous articulations and multiarticular muscles makes assessment of isolated foot and ankle muscle function in patients with CAI very difficult in a clinical setting. A greater understanding of foot and ankle muscle volumes and ankle strength would improve clinical practice by allowing for more informed decisions regarding rehabilitation for patients with lateral ankle sprain and CAI. To fill this gap in knowledge, the purpose of the current investigation was to analyze intrinsic and extrinsic foot and ankle muscle volumes and 4-way ankle strength in young adults with and without CAI.

METHODS

Study Design

A cross-sectional study was performed to compare intrinsic and extrinsic foot and ankle muscle volumes and ankle strength in young adults with and without CAI. Our independent variables were group (CAI or healthy control) and our dependent variables were height × mass-normalized muscle volumes[20] and mass-normalized 4-way ankle strength (normalized force output for dorsiflexion, plantar flexion, inversion, and eversion). The study methods were approved by the University of Virginia's Institutional Review Board, and all subjects provided informed consent prior to study participation.

Participants

Five young adults with CAI and 5 healthy controls volunteered to participate in this study (Table 1). Inclusion criteria for the CAI subjects were a history of more than 1 significant ankle sprain, with the initial sprain occurring more than 1 year prior to study onset, and current self-reported functional deficits due to ankle symptoms that were quantified by a score of <75% on the Foot and Ankle Ability Measure (FAAM) Sport scale and a score of $\geq$10 on the Identification of Functional Ankle Instability scale (IdFAI). Because of the small sample size and exploratory nature of this study, we utilized a lower functional threshold for CAI subjects (<75% on FAAM Sport) than previous investigations (<85% on FAAM Sport).[3,15] Healthy subjects were age-, sex-, and limb-matched (right or left) to the subjects with CAI and were self-reported to be healthy with no history of ankle sprain to either limb. Exclusion criteria for both groups included lower extremity surgery, lower extremity fracture, foot or ankle immobilization greater than 48 hours within 6 months of study onset, or any other condition known to affect muscle volumetric measurements (muscular dystrophy, multiple sclerosis, etc). Subjects with CAI were also excluded if they had an ankle sprain within 6 weeks of study onset, and all subjects were required to be physically active (20 minutes/day at least 3 days/week). None of the subjects had previously completed any supervised rehabilitation for their ankle sprains.

TABLE 1
Subject Demographics[a]

	CAI (n = 5)	Controls (n = 5)
Age, y	23.0 ± 4.0	23.8 ± 4.5
Sex, male:female, n	1:4	1:4
Height, cm	165.4 ± 8.8	166.9 ± 8.1
Mass, kg	66.5 ± 7.3	65.0 ± 13.1
Number of ankle sprains	3.2 ± 1.6	N/A
Time from last sprain, mo	27.8 ± 21.2	N/A
FAAM subscale		
ADL	89.9 ± 3.6	100.0 ± 0.0
Sport	54.4 ± 22.1	100.0 ± 0.0
IdFAI	24.0 ± 3.8	N/A
Godin Leisure-Time Physical Activity Questionnaire	51.8 ± 23.0	65.8 ± 29.1

[a] Data are reported as mean ± SD unless otherwise indicated. ADL, activities of daily living; CAI, chronic ankle instability; FAAM, Foot and Ankle Ability Measure, IdFAI, Identification of Functional Ankle Instability; N/A, not applicable.

Instruments

Magnetic Resonance Imaging for Foot and Ankle Muscle Volumes. Subjects were scanned on a 3-T magnetic resonance imaging (MRI) scanner (Magnetom Trio; Siemens) from just superior of the medial and lateral femoral condyles through the entire foot and ankle. Images were acquired using a 2-dimensional (2D) multislice non-Cartesian spiral gradient echo sequence,[20] and scan time was approximately 15 minutes per subject. Scan parameters for the shank were as follows: echo time/repetition time/alpha (TE/TR/α), 3.8 ms/800 ms/90°; field of view, 400 × 400 mm; slice thickness, 5 mm; in-plane spatial resolution, 1.1 × 1.1 mm. Scan parameters were identical for the foot with the exception of a smaller field of view (250 × 250 mm) and commensurately higher resolution. Because of the smaller field of view for the intrinsic foot muscles, a Siemens 4-channel large flex coil was utilized to increase the signal-to-noise ratio.

Four-Way Ankle Strength Testing. Ankle strength (dorsiflexion, plantar flexion, inversion, and eversion) was measured using a handheld dynamometer (Accelerated Care Plus Corp).

Procedures

Subjects completed a general health history questionnaire, Godin Leisure-Time Physical Activity Questionnaire,[17] FAAM activities of daily living[33] and sport subscales,[6] and IdFAI questionnaire.[11] Prior to strength testing, subjects performed a 5-minute warm-up by walking on a treadmill at a self-selected pace. For each testing position,[29] subjects were instructed to complete practice trials at 50% and then 75% of maximal effort against the tester's resistance. Three 5-second maximal voluntary isometric contractions (MVICs) were completed with a 15-second rest period between trials. All 3 trials for individual ankle motion were completed before transitioning to the 50% and 75% practice trials of the next tested ankle motion. We utilized strength testing positions as recommended for hand-held dynamometry of the lower extremity by Kelln et al.[29] MRI was scheduled within 1 week of strength testing.

Subjects were positioned in the MRI scanner supine and feet first. Axial slices for the shank were obtained contiguously in sets of 20 images from just superior to the femoral condyles distally through the most inferior aspect of the calcaneus. The flex coil was then applied around the feet, and axial slices were obtained in sets of 20 images from just posterior to the calcaneus anteriorly through the entire foot.

Data Reduction

MRI Processing. A detailed and technical description of the data processing technique has been published previously.[20] Briefly, each intrinsic and extrinsic foot and ankle muscle was segmented using in-house segmentation software written in Matlab (Mathworks). Specific muscles segmented are listed in Tables 2 and 3. The segmentation process required the investigator to specify 2D contours, which define the perimeter of each muscle, in each axial slice (Figure 1). The segmentation analysis was performed by 3 trained research assistants who utilized a detailed slice-by-slice segmentation atlas created from a previous data set using similar scanning parameters and segmentation procedures. The research assistants were blinded to group membership during segmentation of all axial slices. The final images were then screened by a single highly trained investigator to ensure consistency across all segmented images. The 2D area of each muscle for each axial slice was multiplied by the slice thickness (5 mm) to obtain the muscle volume for that slice, and the segmentation software created 3-dimensional (3D) in vivo reconstructions of all intrinsic and extrinsic foot and ankle muscles and calculated the associated muscle volumes. Muscle volumes were normalized to each subject's height × mass.[20] Normalized muscle volumes ($cm^3/m{\cdot}kg$) were utilized to compare groups. We compared the individual muscle volumes as well as summed compartmental (anterior, lateral, deep posterior, superficial posterior) and total muscle volume for the extrinsic muscles and total intrinsic plantar muscle volumes between groups. For extrinsic muscle volume comparisons we also compared normalized muscle volumes to normative values of healthy individuals,[20] as described in the Statistical Analysis section.

Four-Way Ankle Strength. Strength was recorded as the maximal force (N) output during the individual MVIC trials for each ankle motion. The average over the 3 trials was computed for each of the 4 tested motions and normalized to each subject's mass (kg), and the normalized force output (N/kg) was utilized to compare groups. Composite ankle strength was calculated from the summed total of the 4 ankle motions and treated as a separate dependent variable.

TABLE 2
Extrinsic Foot and Ankle Muscle Volumes and Associated Cohen *d* Effect Sizes[a]

	Muscle Volume, $cm^3/m{\cdot}kg$		
Extrinsic Muscle	CAI	Controls	Cohen *d* Effect Size
Tibialis anterior	0.92 ± 0.04 (0.90-0.95)	0.97 ± 0.18 (0.84-1.10)	0.36 (–0.69 to 1.41)
Phalangeal extensors	0.63 ± 0.12 (0.53-0.72)	0.72 ± 0.16 (0.60-0.84)	0.65 (–0.41 to 1.72)
Peroneals	0.91 ± 0.15 (0.80-1.02)	0.94 ± 0.17 (0.82-1.07)	0.21 (–0.83 to 1.25)
Flexor digitorum longus	0.16 ± 0.02 (0.15-0.18)	0.18 ± 0.04 (0.15-0.21)	0.48 (–0.58 to 1.53)
Flexor hallucis longus	0.87 ± 0.22 (0.71-1.02)	1.05 ± 0.32 (0.82-1.29)	0.68 (–0.39 to 1.75)
Tibialis posterior	0.86 ± 0.09 (0.79-0.92)	0.89 ± 0.11 (0.82-0.97)	0.38 (–0.67 to 1.43)
Popliteus	0.13 ± 0.01 (0.12-0.14)	0.16 ± 0.04 (0.13-0.19)	1.18 (0.05 to 2.30)
Gastrocnemius medial head	1.62 ± 0.17 (1.50-1.75)	1.93 ± 0.29 (1.73-2.14)	1.30 (0.15 to 2.44)
Gastrocnemius lateral head	0.90 ± 0.17 (0.78-1.03)	1.02 ± 0.16 (0.93-1.11)	0.69 (–0.38 to 1.76)
Soleus	2.62 ± 0.30[b] (2.40-2.84)	3.26 ± 0.54[b] (2.86-3.66)	1.46 (0.29 to 2.63)

[a] Data are reported as mean ± SD unless otherwise indicated; values in parentheses represent 90% CIs. Positive effect size indicates lower muscle volumes with chronic ankle instability (CAI).

[b] Significant difference as indicated by group means and associated 90% CIs that do not overlap.

Statistical Analysis

All dependent variables (muscle volume and strength) were compared between groups (CAI and healthy control) with group means and associated 90% CIs. For dependent variables where the CIs between groups did not overlap, it was determined that the groups were significantly different. We also calculated Cohen *d* effect sizes and associated 90% CIs to estimate the magnitude and precision of group differences. Effect sizes were interpreted as follows: ≥0.80, large; 0.50 to 0.79, moderate, 0.20 to 0.49, small; and <0.20, trivial.[7] Given the novelty of this study, we also utilized the smallest significant difference from our current results (where 90% CIs did not overlap between groups) to estimate the sample size that would be required per group to find significance at an alpha level (type I error) of 0.05 and power ($1 - \beta$) of 0.8. Data were analyzed using Microsoft Excel Version 14.1.0 (Microsoft).

TABLE 3
Intrinsic Foot Muscle Volumes and Associated Cohen *d* Effect Sizes[a]

Intrinsic Foot Muscle	Muscle Volume, cm^3/m·kg CAI	Muscle Volume, cm^3/m·kg Controls	Cohen *d* Effect Size
Abductor hallucis	0.21 ± 0.06 (0.17-0.25)	0.21 ± 0.05 (0.17-0.25)	0.06 (–0.98 to 1.10)
Adductor hallucis obliquus	0.07 ± 0.01[b] (0.06-0.08)	0.13 ± 0.03[b] (0.11-0.15)	2.33 (0.98 to 3.68)
Adductor hallucis transversus	0.02 ± 0.008 (0.01-0.02)	0.02 ± 0.008 (0.01-0.02)	–0.19 (–1.23 to 0.85)
Flexor hallucis brevis	0.06 ± 0.02[b] (0.05-0.07)	0.12 ± 0.02[b] (0.11-0.14)	3.42 (1.79 to 5.06)
Abductor digiti minimi	0.16 ± 0.02 (0.14-0.18)	0.17 ± 0.03 (0.14-0.19)	0.13 (–0.91 to 1.17)
Flexor digiti minimi	0.08 ± 0.03 (0.06-0.10)	0.06 ± 0.01 (0.05-0.07)	–0.60 (–1.67 to 0.46)
Extensor digitorum brevis	0.08 ± 0.03 (0.06-0.10)	0.11 ± 0.04 (0.07-0.14)	0.70 (–0.38 to 1.77)
Flexor digitorum brevis	0.20 ± 0.05 (0.16-0.23)	0.22 ± 0.05 (0.18-0.25)	0.37 (–0.68 to 1.42)
Interosseus	0.17 ± 0.04 (0.14-0.20)	0.19 ± 0.03 (0.17-0.21)	0.45 (–0.60 to 1.50)
Quadratus plantae	0.13 ± 0.04 (0.10-0.16)	0.14 ± 0.02 (0.12-0.15)	0.28 (–0.77 to 1.33)
Total plantar intrinsic foot muscle volume	1.09 ± 0.19 (0.95-1.23)	1.24 ± 0.15 (1.13-1.35)	0.88 (–0.21 to 1.97)

[a] Data are reported as mean ± SD unless otherwise indicated; values in parentheses represent 90% CIs. Positive effect size indicates lower muscle volumes with chronic ankle instability (CAI).

[b] Significant difference as indicated by group means and associated 90% CIs that do not overlap.

Normative Database Comparison. We also compared extrinsic muscle volumes to a previously established normative database for lower extremity muscle volumes.[20] The database was created as part of another project that quantified the relationship between lower extremity muscle volumes to body mass and height in 24 healthy subjects.[20] To compare CAI subjects in our current study to the previously published normative values,[20] we calculated z scores for each extrinsic muscle, individually for all 5 CAI subjects; z scores were calculated to describe how many standard deviations the muscle volumes of CAI subjects were from the normative database mean muscle volume using the following formula:

$$\frac{\textit{CAI Subject Muscle Volume} - \textit{Normative Database Mean Muscle Volume}}{\textit{Normative Database Standard Deviation}}$$

For descriptive purposes, we also provide the average z scores of the 5 healthy controls in our study to compare with previously published normative values. Our current study is one of the first studies to quantify the intrinsic foot muscle volumes using this technique, and thus, it was not possible to compare the intrinsic foot muscles to normative values. Clinical interpretation of z scores was determined a priori as follows: $z \geq 3.0$, extreme hypertrophy; $3 > z \geq 2$, moderate hypertrophy; $2 > z \geq 1$, slight hypertrophy; $1 > z > -1$, normal; $-1 \geq z > -2$, slight atrophy; $-2 \geq z > -3$, moderate atrophy; and $-3 \geq z$, extreme atrophy.

RESULTS

Muscle Volumes

Extrinsic Foot and Ankle Muscle Volume: Group Comparisons. The CAI group presented smaller total extrinsic foot and ankle muscle volumes compared with age- and sex-matched healthy controls (CAI, 9.62 ± 0.39 cm^3/m·kg; healthy, 11.13 ± 1.33 cm^3/m·kg). Additionally, the CAI group had smaller summed muscle volume of the superficial posterior compartment (CAI, 5.15 ± 0.55 cm^3/m·kg; healthy, 6.21 ± 0.73 cm^3/m·kg) (Figure 2). Individual CAI muscle volume deficits were identified for the soleus muscle (CAI, 2.62 ± 0.30 cm^3/m·kg; healthy, 3.26 ± 0.54 cm^3/m·kg) (Table 2). Effect sizes for the total muscle volume, superficial posterior compartment, and soleus muscle were all large and ranged from 1.46 to 1.63 with confidence intervals that did not cross zero. No other significant differences were identified for extrinsic foot and ankle muscle volume comparisons between groups.

Extrinsic Foot and Ankle CAI Muscle Volume: Normative Database Comparisons. CAI subjects presented slight atrophy (mean z score, $-1 \geq z > -2$) of the flexor digitorum longus (mean z, –1.23) and soleus (mean z, –1.45) when compared with the normative database (Figure 3). Subjects with CAI also demonstrated moderate atrophy (mean z, $-2 \geq z > -3$) of the medial gastrocnemius (mean z, –2.00), lateral gastrocnemius (mean z, –2.16), phalangeal extensors (mean z, –2.06), and the popliteus (mean z, –2.64) but moderate hypertrophy of the flexor hallucis longus (mean z, 1.88) when compared with the normative database. Nine of 10 extrinsic foot and ankle muscle volume z scores for the healthy controls fell within 1.15 standard deviations (–1.15 < healthy control z scores < 1.15) when compared with normative database values. The healthy controls presented with a mean z score of –1.41 for the popliteus muscle when compared with the normative database.

Intrinsic Foot and Ankle Muscle Volumes: Group Comparisons. The CAI group presented smaller muscle volumes for the adductor hallucis obliqus (CAI, 0.07 ± 0.01cm^3/m·kg; healthy, 0.13 ± 0.03 cm^3/m·kg) and flexor hallucis brevis (CAI, 0.06 ± 0.02 cm^3/m·kg; healthy, 0.12 ± 0.02 cm^3/m·kg) (Table 3). Effect sizes were 2.33 for the adductor hallucis obliqus and 3.42 for the flexor hallucis longus, both with CIs that did not cross zero, suggesting large muscle volume deficits for these 2 intrinsic foot muscles. There were no other significant differences for individual intrinsic foot muscle volumes or for the summed total plantar intrinsic foot muscle volume (Table 3).

Four-Way Ankle Strength

The CAI group demonstrated significantly less force output for dorsiflexion (CAI, 1.97 ± 0.23 N/kg; healthy,

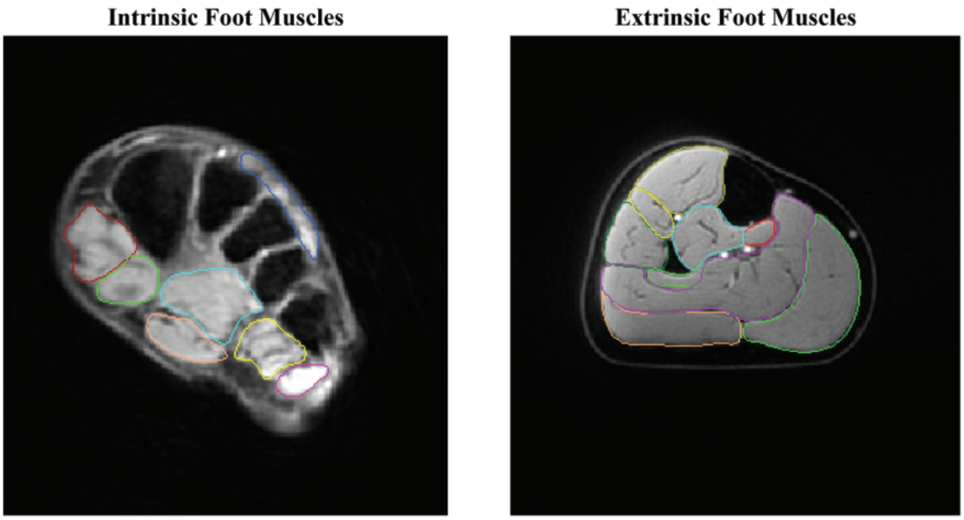

Figure 1. Segmented axial slice of the intrinsic (left) and extrinsic (right) foot and ankle muscles.

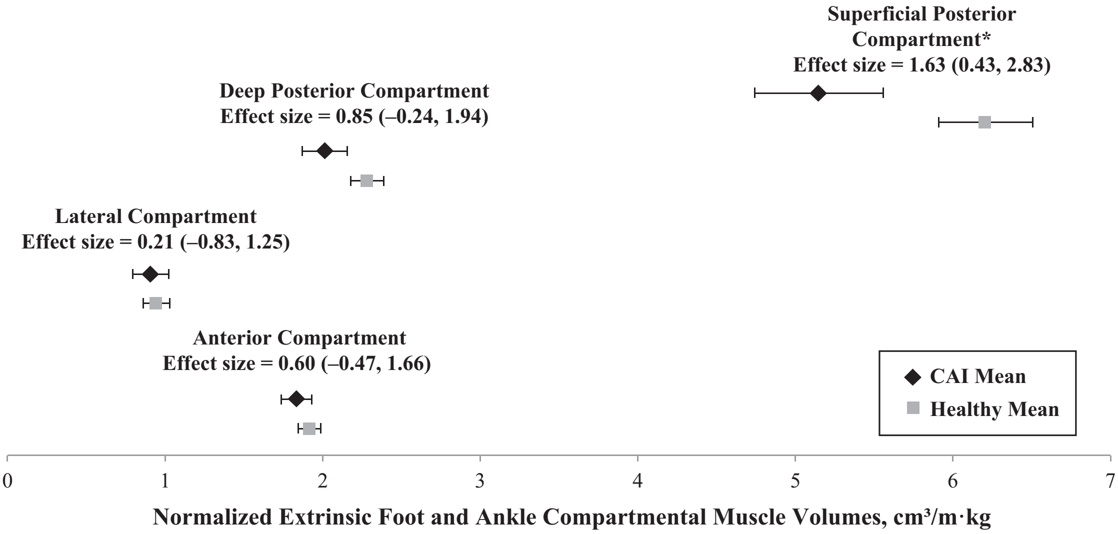

Figure 2. Extrinsic foot and ankle muscle compartment volumes ($cm^3/m{\cdot}kg$) and associated Cohen *d* effect sizes and 90% CIs. *Significant difference as indicated by group means and associated 90% CIs that do not overlap. Positive effect size indicates lower muscle volumes with chronic ankle instability (CAI).

2.67 ± 0.47 N/kg), eversion (CAI, 1.82 ± 0.27 N/kg; healthy, 2.39 ± 0.30 N/kg), and total composite strength measures (CAI, 9.12 ± 1.18 N/kg; healthy, 11.48 ± 1.64 N/kg). Effect sizes for significant group differences in strength were all large effect sizes that ranged from 1.66 to 2.03, with CIs that did not cross zero. The CAI group also had lower, but not statistically significant, inversion (CAI, 1.54 ± 0.31 N/kg; healthy, 2.05 ± 0.41 N/kg) and plantar flexion (CAI, 3.79 ± 0.67 N/kg; healthy, 4.37 ± 0.69 N/kg) strength with effect sizes of 1.43 and 0.84, respectively.

Sample Size Estimate

We performed a sample size estimation from the current data based on a minimal group difference of 0.64 $cm^3/m{\cdot}kg$ and variability of 0.42 $cm^3/m{\cdot}kg$ in the soleus muscle. We estimated that we would need 7 subjects per group to find significant differences at an alpha level of 0.05 and power of 0.8.

DISCUSSION

We identified large deficits in muscle volume and concurrent 4-way ankle strength weakness in CAI patients when compared with age-, sex-, and limb-matched healthy controls. To our knowledge, this is the first study to quantify and compare muscle volumes of the intrinsic and extrinsic foot and ankle muscles between young adults with and without CAI. This novel investigation into CAI muscle morphology should provide insight to sports medicine clinicians

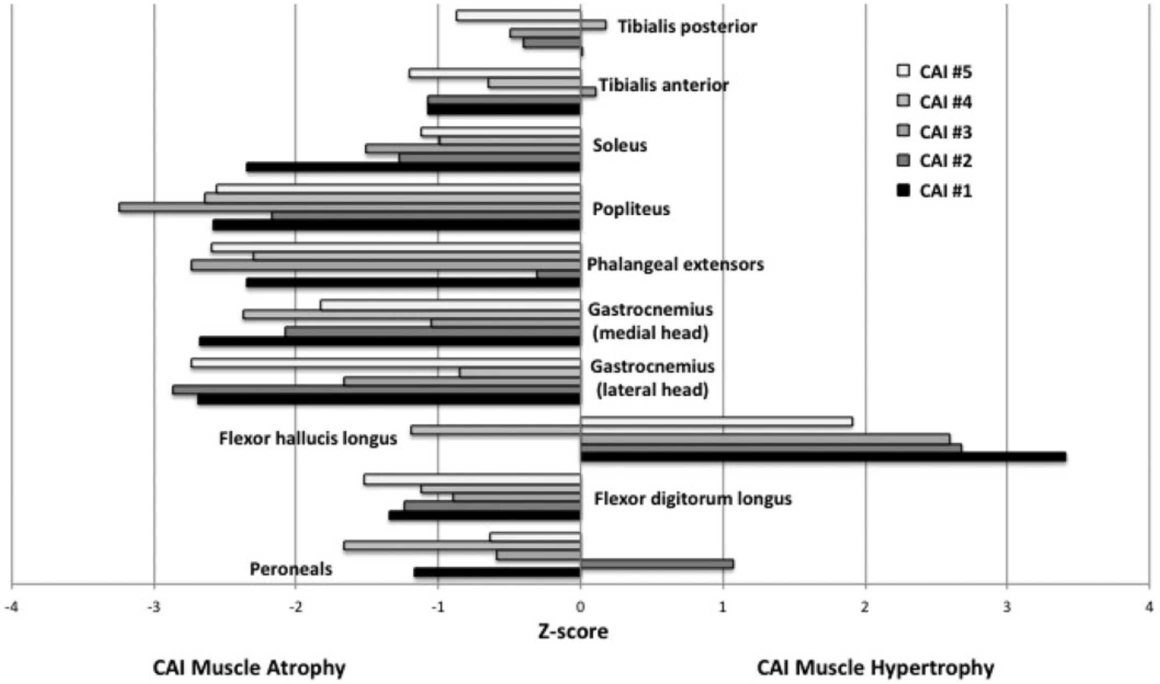

Figure 3. Extrinsic muscle volume normative database comparisons (*z* scores) for each individual subject with chronic ankle instability (CAI).

and researchers involved with the assessment and treatment of muscle strength and function following lateral ankle sprain and in patients with CAI.

Patients with CAI exhibited large deficits in total extrinsic muscle volume, which was driven by the large group differences in the superficial posterior compartment. For individual extrinsic muscles, patients with CAI demonstrated significantly smaller soleus muscle volume. When compared with the normative database,[20] we confirmed the CAI soleus muscle volume deficit and elucidated potential muscle volume deficits of the flexor digitorum longus, medial and lateral gastrocnemius, phalangeal extensors, and popliteus muscles. The normative database comparison also identified potential hypertrophy of the flexor hallucis longus in the CAI group.

As expected, smaller summative extrinsic foot and ankle muscle volumes with CAI were accompanied by large deficits in the total 4-way composite ankle strength, which is consistent with the existing literature.[23,26] It should be noted that subjects with CAI were slightly less physically active than the healthy controls in this study, and this is also consistent with existing literature.[27] However, it is important to consider that the decreased physical activity associated with CAI could exacerbate/contribute to the muscle atrophy and decreased strength seen with CAI. Interestingly, we did not identify any muscle volume differences between groups in the peroneal muscles (lateral compartment). This finding is particularly interesting considering that the CAI eversion strength deficit in our study was the largest group difference, based on effect size, with regard to ankle strength. This suggests the current and previously reported[1] eversion strength deficits may be related to neuromuscular mechanisms as opposed to muscle size and associated torque-generating capacity. We can only speculate about whether the neuromuscular mechanisms are due to supraspinal, spinal, or peripheral mechanisms[21] that result in disproportional force output for a similar muscle volume; however, it does not appear from our results that eversion strength deficits are driven by muscle atrophy of the peroneals. Peroneus longus neuromuscular dysfunction has been reported during gait[14,42] and in response to inversion perturbations[25,30,37]; however, our current results suggest that neuromuscular dysfunction may also play a role during assessment of eversion ankle strength in patients with CAI.

Inversion and dorsiflexion strength deficits have been previously reported in patients with CAI,[23,26,28] and our strength results corroborate these findings. Unlike our results for the peroneal muscles, the concurrent muscle volume analysis suggests that inversion and dorsiflexion strength deficits are, in part, related to moderate deficits in the anterior compartment muscle volume (anterior tibialis and phalangeal extensors) with CAI. Clinicians may benefit from utilizing inversion and dorsiflexion strengthening exercises with parameters of sets and repetitions focused on muscle hypertrophy. Improvements in inversion and dorsiflexion strength have been reported previously in the treatment of patients with CAI[18,19]; however, we do not know whether the improvements in strength seen in those studies were related to muscle hypertrophy or improved neuromuscular function. It should also be noted that there was a fair amount of intersubject variability seen across CAI subjects in our *z* score analysis (see Figure 3) where some subjects had more severe atrophy of some muscles and less severe atrophy of other muscles. This finding underscores the multifaceted nature of CAI and how each patient will need an individualized approach to rehabilitation, as suggested by Donovan and Hertel.[12]

Posttraumatic ankle osteoarthritis has been shown to be a long-term consequence of repetitive ankle sprain and CAI.[39,43] Patients with posttraumatic osteoarthritis demonstrate soleus muscle atrophy,[44] and we have now identified that young adults with CAI, without a clinical diagnosis of osteoarthritis, also demonstrate soleus muscle atrophy. However, the collective plantar flexor muscle atrophy in the superficial posterior compartment did not translate into significantly lower plantar flexion strength in our analysis. Previous studies with larger cohorts have been able to demonstrate plantar flexion deficits in patients with CAI.[16] Additionally, the large effect size demonstrating a potential plantar flexion weakness with CAI in our study suggests that the study was likely underpowered to detect this group difference. However, it is also possible that the moderate hypertrophy, seen in the flexor hallucis longus in 4 of 5 patients with CAI, muted the plantar flexion deficit that might have been expected from the smaller superficial posterior compartment.

The flexor hallucis longus has been described as a first ray plantar flexor and stabilizer of the medial longitudinal arch during gait.[41] Numerous studies have analyzed gait pathomechanics in patients with CAI, and heavy emphasis has been placed on rearfoot mechanics and peroneal dysfunction due to the mechanism of ankle sprain and the ability of the peroneal muscles to counteract inversion moments. However, we posit that our finding of hypertrophy of the flexor hallucis longus and atrophy of the flexor hallucis brevis and flexor hallucis obliqus may necessitate future investigation into the role of the segmental motion of the first ray during gait and other functional tasks in patients with CAI. This hypothesis is supported by previous studies that demonstrate a relationship between first ray rigidity and increased lateral plantar pressure[5] that interestingly coincides with the characteristic gait pattern in patients with CAI.[35,36,40] The laterally deviated plantar pressure seen with CAI[35,36,40] could increase the load on the flexor hallucis longus while stabilizing the supinated foot and medial longitudinal arch, potentially resulting in hypertrophy of the flexor hallucis longus over time. Similarly, decreased first ray excursion and decreased force transmitted through the first ray could limit the stress placed on the flexor hallucis brevis and flexor hallucis obliqus, potentially resulting is disuse atrophy. These hypotheses about first ray gait pathomechanics are speculative and should be investigated further in individuals with and without CAI.

Limitations

Results from the present study represent only 5 patients with CAI, and the results may not be generalizable to individuals with a lower degree of self-reported disability. The small sample size for this novel investigation was due to the large amount of data processing time required (approximately 12 hours per subject); however, our estimated sample size indicates that the large group differences identified are likely meaningful differences in muscle volume with CAI. Future advances toward automatic segmentation of muscles may reduce this time and promote studies with larger populations. Strength measurements with handheld dynamometers do not adequately approximate the dynamic nature of muscle contraction or function needed to prevent ankle sprain and the muscle weakness identified in our study should not be interpreted in a cause-effect relationship with recurrent sprain. Additionally, the lack of electromyographic measures from the foot and ankle muscles prevented us from directly linking neuromuscular dysfunction to the diminished muscle volume and strength measures seen in the CAI group. Future work involving electromyography will serve as an interesting complement to the present work.

CONCLUSION

We identified large deficits in intrinsic and extrinsic foot and ankle muscle volumes in young adults with CAI when compared with age-, sex-, and limb-matched healthy counterparts. These results elucidate the pathophysiology of CAI, and clinicians should be aware of the potential foot and ankle muscle volume deficits when prescribing rehabilitation for patients with a history of repetitive ankle sprain.

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