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Acquired midfoot deformity and function in individuals with diabetes and peripheral neuropathy

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Abstract

Background—Diabetes mellitus related medial column foot deformity is a major contributor to ulceration and amputation. However, little is known about the relationship between medial column alignment and function and the integrity of the soft tissues that support and move the medial column. The purposes of this study were to determine the predictors of medial column alignment and function in people with diabetes and peripheral neuropathy.

Methods—23 participants with *diabetes and neuropathy* had radiographs, heel rise kinematics, magnetic resonance imaging and isokinetic muscle testing to measure: 1) medial column alignment (Meary's angle- the angle between the 1st metatarsal longitudinal axis and the talar head and neck), 2) medial column function (forefoot relative to hindfoot plantarflexion during heel rise), 3) intrinsic foot muscle and fat volume, ratio of posterior tibialis to flexor digitorum tendon volume, 4) plantar fascia function (Meary's angle change from toes flat to extended) and 5) plantarflexor peak torque. Predictors of medial column alignment and function were determined using simultaneous entry multiple regression.

Findings—Posterior tibialis to flexor digitorum tendon volume ratio and intrinsic foot muscle volume were significant predictors of medial column alignment ($p<.05$), accounting for 44% of

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the variance. Intrinsic foot fat volume and plantarflexor peak torque were significant predictors of medial column function ($p<.05$), accounting for 37% of the variance.

Interpretation—Deterioration of medial column supporting structures predicted alignment and function. Prospective research is required to monitor alignment, structure, and function over time to inform early intervention strategies to prevent deformity, ulceration, and amputation.

Keywords

Intrinsic foot muscle; plantarflexor power; posterior tibialis tendon; muscle volume

1. Introduction

Adult-acquired neuropathic mid foot deformity in individuals with diabetes mellitus (DM) is a major contributor to high plantar pressure that often leads to ulceration and ultimately lower extremity amputation.[1] Lower extremity amputations are associated with high rates of morbidity and mortality.[2-4] Deformities that occur along the medial column of the foot (calcaneus, talus, navicular, cuneiforms, and metatarsals 1 through 3) are particularly devastating. Medial column deformities are difficult to accommodate in footwear, often resulting in inadequate offloading of plantar pressure to allow healing or maintenance of skin integrity. Understanding the factors that contribute to acquired neuropathic medial column foot deformity is critical in order to halt or prevent the cascade of events that will decrease the quality and shorten the lives of the more than 380 million people worldwide that have DM. [5]

Medial column foot alignment and function are, in part, determined by intrinsic and extrinsic muscular and fascia support.[6] Loss of intrinsic foot muscle volume and infiltration with fat has consistently been found in individuals with DM and peripheral neuropathy (PN)[7, 8] and intrinsic foot muscle deterioration has been associated with severity of the metatarsophalangeal joint angle.[8] Up to a 21% decrease in strength measures of extrinsic ankle plantarflexor muscles has been measured in people with DM compared to age matched controls.[9-11] In addition, posterior tibialis tendon dysfunction is often implicated in development of medial column deformity in adults without DMPN.[12-15] The plantar fascia has been found to be thickened in individuals with DM and there is evidence to support an impaired ability of the plantar fascia to raise the arch when toes are extended in individuals with midfoot deformity.[16-18]

Bony alignment of the medial column of the foot is also thought to be dependent on how the foot moves during daily activities. Limited ankle dorsiflexion can result in a compensatory pattern requiring excessive motion particularly at mid tarsal joints. Ankle dorsiflexion range of motion has been found to be limited in those with DMPN and limited ankle dorsiflexion has been found to be related to acquired flat foot deformity in those with posterior tibialis tendon dysfunction .[19-23] We have also identified that some individuals with DM have limited ability to plantarflex their forefoot on their hindfoot during the heel rise task, potentially compromising midfoot joint stability during loading.[24]

Prevention of medial column deformity and maintenance of normal midfoot function during daily activities are paramount in reducing the ulceration and amputation rates in those with DM. Identification of the key predictors of medial column deformity and function would assist in developing appropriate prevention and treatment strategies. The primary purposes of this study were to determine the predictors of medial column alignment and medial column function in people with DMPN. We hypothesized that the key predictors of medial column alignment (i.e. Meary's angle) would be intrinsic foot muscle deterioration,[8] posterior tibialis tendon volume, plantar fascia function,[25] and ankle dorsiflexion. We hypothesized that the key predictors of midfoot function (i.e. forefoot relative to hindfoot plantarflexion excursion during heel rise)[24] would be intrinsic muscle deterioration,[8] posterior tibialis tendon volume, extrinsic plantarflexor muscle strength,[26] and plantar fascia function[25].

2. Methods

2.1 Participants

Twenty-three participants with DM, PN, and a range in severity of midfoot deformity were recruited from diabetes clinics and volunteer databases. Twelve age- and weight-matched participants without DM, PN, and midfoot deformity were also tested to provide comparative data for all measures. All participants were informed of the study requirements and signed a consent form approved by our Institutional Review Board prior to study participation. The DMPN group had either type 1 or type 2 DM, diagnosed by the participants' physician and requiring either oral hypoglycemic or exogenous insulin medication for blood glucose control. Peripheral neuropathy was determined by the inability to feel the 5.07 (10-gram) Semmes-Weinstein monofilament on a minimum of one of six locations on the plantar foot surface.[27] Study exclusion criteria for all groups were: 1) lower extremity amputations that extended beyond the toes, 2) current plantar ulcer, 3) non-ambulatory, or 4) weighed >180 kg, or had metal implants or pace makers (restrictions associated with magnetic resonance imaging).

2.2 Deformity

The presence and severity of medial column deformity was measured using the following clinical measures and criteria: 1) standing calcaneal eversion relative to the leg (deformity considered present if ≥5°),[28] 2) medial longitudinal arch angle (angle between the medial malleolus, navicular, and 1st metatarsal head in standing, deformity considered present if <130°),[28, 29] 3) height of the most inferior part of the navicular to the ground (deformity considered present if ≤24 mm),[30] 4) arch index (standing dorsal foot height/truncated foot length, deformity considered present if <0.263),[31] and 5) medial midfoot peak plantar pressure during barefoot walking measured by EMED-ST P-2 pressure platform (Novel Inc., St. Paul, MN) (deformity considered present if >290 kPa).[31] Control participants could meet ≤ 1 of the deformity criteria above to be included in the study. For DMPN and control participants who met ≤1 of the criteria, the foot studied was the one that had the least signs of medial column deformity. For DMPN participants who met >1 of the criteria, the foot studied was the one that had the most signs of medial column deformity. This strategy provided a DMPN group with a large range in severity of medial column alignment.

Medial column alignment was quantified by measuring Meary's angle (the angle formed by the line that bisects the talar head and neck and the line through the longitudinal axis of the 1st metatarsal) from lateral weightbearing radiographs. Methods used to analyze the foot radiographs have been previously described in detail.[25] In short, the lateral radiographic images were imported into iSite® Picture Archiving and Communication Systems (PACS) software (Philips Healthcare Informatics, Foster City, CA). A single experienced rater, who was blinded to the group assignment, measured all radiographs.

2.3 Posterior lower leg and foot muscle, fat, tendon, and fascial volume

Magnetic resonance images (MRI) of the involved side calf and foot were acquired using a Siemens Magnetom Trio 3T MRI scanner (Siemens Medical Systems, Malvern, PA). MR parameters are described in the supplementary material. (Supplementary table) The measurement methods for calf and foot muscle and fat are reported in detail in previous publications.[32, 33] Briefly, calf muscle and fat images were acquired while the participant lay supine with a Siemens extremity coil surrounding the involved leg. Images with and without fat saturation were collected beginning at the tibiofemoral joint and continued distal. Images were loaded into Analyze (Biomedical Imaging Resource, Mayo Clinic, Rochester, MN) where nine consecutive slices, 11 slices distal from the joint line, were selected for volume measurement. The lean muscle and intermuscular adipose tissue volumes were segmented and measured for the gastrocnemius, soleus, and posterior deep compartments. The foot images were collected in supine with the foot placed inside the head coil. A set of coronal images were collect from the posterior calcaneus toward the toes. Intrinsic muscle and fat volumes were measured for all slices from the talonavicular joint to the tarsometatarsal joint.[8]

A set of transverse images were collected, perpendicular to the posterior tibialis, just distal of the talocrural joint progressing proximal. Achilles, posterior tibialis, flexor digitorum, and flexor halluces longus tendons volumes were measured starting at the talocrural joint space continuing superior for 9 slices. Tendon volumes were measured using a voxel-based image segmentation method called "region growing" using Analyze software (Mayo Clinic Biomedical Imaging Resource, Rochester, MN).[34] In MR images, tendons are dark structures surrounded by lighter voxel intensity structures (tendon sheaths, fat etc). The "region growing" function uses this difference in voxel intensities to define the borders of the tendon. The user places an initial "seed" point within the dark tendon to determine the starting intensity value. As the user changes the intensity threshold, the algorithm evaluates voxels adjacent to the seed and adds the voxels that are within the threshold until the borders of the region of interest best match the borders of the tendon.(Figure 1*)* A ratio of posterior tibialis tendon to flexor digitorum longus tendon volume was calculated for each participant. Radiologist commonly use this method of "normalizing" tendon size for a particular individual and expect the posterior tibialis tendon to be twice the size of the flexor digitorum longus tendon.[35] All leg and foot muscle, and tendon volumes were measured by a single rater (initials removed for blinding). This single rater's, test-retest reliability (2 weeks between measures) was examined for the tendon and plantar fascia volume measures and showed good to excellent agreement [ICC(3,1): flexor digitorum longus (0.91), flexor hallucis longus (0.94), Achilles (0.97) and posterior tibialis (0.98)].

2.4 Plantarflexor strength

Concentric plantarflexor torque at 60 degrees/sec was assessed using the Biodex System 3 Pro Orthopedic Testing & Rehabilitation dynamometer (Biodex Medical Systems, 20 Ramsay Rd, Shirley, New York 11967-4704, USA). Three trials, each with 3 repetitions, were completed. Strength (peak torque) was calculated as the average of the two highest torque values across trials.

2.5 Lower extremity kinematics

Heel rise kinematic and kinetic data for the shank, hindfoot, and forefoot were captured with an 8-camera, 200 Hz Vicon motion analysis system (Vicon MX, Los Angeles, California, USA) and a Bertec K80301 force platform (Bertec Corporation, Columbus, OH, USA). The shank markers were placed on the fibular head, tibial tuberosity, medial and lateral malleoli, and a four marker plate at the lateral distal tibia. The hindfoot markers were located at the sustenaculum tali, peroneal trochlea and a two marker plate that bisected the posterior calcaneus. The forefoot markers were at the head and base of the 1st and 5th metatarsals and one between the 2nd and 3rd metatarsal heads. The models were built in Visual3D software (C-Motion Inc., Germantown, MD, USA) using a modification of the Oxford foot model detailed in the supplementary material from our previous publications.[36] The participants were instructed to perform up to 20 single leg heel rises. Three trials with the highest plantarflexor power production were selected for analysis. The variable of interest was excursion of forefoot relative to hindfoot plantarflexion from initial to peak height of the heel rise task and used as an indication of midfoot function. The data for the heel rise task in this group of participants have been reported in detail in previous publications.[24]

2.6 Plantar fascia function

Plantar fascia function was measured as the change in Meary's angle (described in section 2.2) from a the lateral, weightbearing foot radiographs with the toes flat and with the toes extended on a board fixed at a 60-degree angle.[25]

2.7 Statistical analyses

Participant characteristics between groups were compared using a two-tailed independent t-test for continuous variables and Chi-square test for discrete variables.

For this study we aimed to identify the predictors of medial column foot alignment (Meary's angle measured from standing, toe flat radiographs) and foot function (forefoot relative to hindfoot plantarflexion excursion during the heel rise task). Simultaneous entry multiple regression analysis was chosen because it is a more conservative approach, appropriate when there is no a priori hypothesis determining the order and importance of predictor variable entry. The following steps were followed in the simultaneous entry multiple regression analysis:

1) A priori hypotheses:

- **a.** Meary's Angle: We hypothesized the medial column foot alignment would be predicted by posterior tibialis tendon/flexor digitorum longus

volume ratio, intrinsic muscle or fat volume, plantar fascia function, and ankle dorsiflexion range of motion.

b. Forefoot relative to hindfoot plantarflexion excursion during heel rise: We hypothesized the ability to plantarflex the forefoot on the hindfoot would be predicted by a measure of intrinsic muscle or fat volume, posterior tibialis tendon/flexor digitorum longus volume ratio, extrinsic plantarflexor muscle strength, and plantar fascia function.

2) Pearson product-moment correlations between the dependent variables and the potential predictor variables and intercorrelations were examined when a predictor variable was measured in multiple ways or there was potential for intercorrelation between the predictor variables (e.g. intrinsic foot compartment muscle volume or fat volume). For variables that were highly intercorrelated the predictor variable with the highest correlation to the dependent variable was entered into the model.

3) Predictor variables were entered into the model.

Group differences in muscle, fat, tendon, fascial volumes, strength variables, heel rise kinematics, and radiographic measures were analyzed using a two-tailed independent t-test. SPSS Statistics version 21 was used for all statistical analyses (SPSS Statistics Inc., Chicago, USA). Statistical significance was set at $p<.05$. Measurements within this article will be given as mean (standard deviation).

3. Results

3.1 Participant characteristics

DMPN participants were 59 (10) years old and controls were 57(14) years old. For both groups there were more males than females and the mean body mass index indicates class II obesity. The DMPN group had primarily type 2 diabetes with disease duration of 17 years. (Table 1) Navicular height was lower in the DMPN group compared to controls (DMPN=31 (10), Control=38 (5) mm, p= .04). Calcaneal eversion (DMPN=5 (5), Control=4 (4) degrees), medial longitudinal arch angle (DMPN=137 (17), Control=145 (8) degrees), arch index (DMPN=0.316 (0.066), Control= 0.344 (0.0311)), and medial midfoot peak plantar pressure (DMPN=178 (227), Control= 69 (65) kPa) were not different between groups ($p>.05$). (Table 1)

3.2 Predictors of Meary's Angle

Posterior tibialis to flexor digitorum longus tendon volume ratio had a moderately strong inverse correlation with Meary's angle ($r = -0.51$, $p = 0.01$). (Figure 2) Intrinsic foot muscle volume was correlated with Meary's angle ($r = 0.49$, $p = 0.01$) while intrinsic foot fat volume was not. Intrinsic foot muscle volume was included as a model variable. Change in Meary's angle from toe flat to toe extended and ankle dorsiflexion range of motion were not correlated with Meary's angle ($r = -0.07$, $p = 0.76$ and $r = -0.02$, $p = 0.94$, respectively) but were included in the model given our *a priori* hypothesis. (Table 2)

The model, which included posterior tibialis/flexor digitorum longus tendon volume ratio, intrinsic foot muscle volume, plantar fascia function, and ankle dorsiflexion range of motion with the knee extended, predicted 44% of Meary's angle. An increase in posterior tibialis to flexor digitorum longus tendon volume ratio was a significant predictor of a decrease in Meary's angle, accounting for 16% of the variance. A decrease in intrinsic muscle volume was a significant predictor of a decrease in Meary's angle, accounting for 17% of the variance. (Table 3)

3.7 Predictors of forefoot relative to hindfoot plantarflexion excursion

Both intrinsic foot muscle and fat compartment volumes were not significantly correlated with forefoot relative to hindfoot plantarflexion excursion ($r = 0.23$ and $r = -0.35$, respectively). Intrinsic foot fat volume was chosen for the regression model because of the stronger bivariate correlation. The ratio of posterior tibialis to flexor digitorum tendon volume was not correlated with forefoot relative to hindfoot plantarflexion excursion ($r = 0.08$, $p = 0.74$) but was included in the model given our *a priori* hypothesis. Plantarflexor torque and change in Meary's angle from toe flat to toe extended were moderately correlated with forefoot relative to hindfoot plantarflexion ($r=0.40$, $p = .05$ and $r = 0.34$, $p = .10$, respectively). (Table 2)

The model, which included intrinsic foot fat volume, ratio of posterior tibialis to flexor digitorum tendon volume, plantarflexor torque, and change in Meary's angle from toe flat to toe extended, predicted 44% of the excursion of forefoot relative to hindfoot plantarflexion during the heel rise task. An increase in intrinsic muscle fat predicted a decrease in forefoot relative to hindfoot excursion and accounted for 19% of the variance. A decrease in plantarflexor torque predicted a decrease in forefoot relative to hindfoot excursion and accounted for 24% of the variance. (Table 4)

3.2 Muscle, fat, tendon, and plantar fascia volumes

Muscle volumes in the posterior compartment of the leg were all less in the DMPN group compared to controls and the difference was significant for the gastrocnemius muscle ($p<.01$). (Table 2) Although fat volumes in the posterior compartment of the leg were greater in the DMPN group compared to the control group the difference was not significant.

There were no significant differences in tendon volumes between groups. (Table 2)

3.3 Plantarflexor torque and correlations to muscle and fat volumes

Plantarflexor torque was less in the DMPN group compared to controls ($p<.01$) and correlated with muscle volumes of the deep and gastrocnemius compartments ($r = .42$ and $r = .47$ respectively, $p<.01$). Plantarflexor torque was not correlated with fat volume.

3.4 Kinematic and plantar fascia function

Forefoot relative to hindfoot plantarflexion excursion during single-limb heel rise task was less in the DMPN group compared to controls ($p<.01$). There was no group differences in the elevation of Meary's angle from toe flat to toe extended. (Table 2)

4. Discussion

This study provides preliminary evidence that muscle and tendon deterioration are predictive of medial column foot deformity and poor midfoot function in individuals with DMPN. A higher ratio of posterior tibialis to flexor digitorum longus tendon volume, indicating posterior tibialis tendon pathology, and lower intrinsic foot muscle volume were significant predictors of medial column deformity (Meary's angle). In addition, deterioration in plantarflexor torque production and greater intrinsic foot compartment fat were significant predictors of midfoot dysfunction as demonstrated by decreased forefoot on hindfoot plantarflexion excursion.

Our model predicted 44% of the variance in medial column alignment and identified intrinsic muscle and posterior tibialis tendon pathology as the key predictors. Although previous work has identified a number of pathologies in the feet of individuals with diabetes, to our knowledge the relationship of the pathologies to medial column deformity has not been previously explored. [37-39] The presence of sensory neuropathy, preventing early detection of injury and timely treatment, increases the risk that minor tissue injury can progress to deformity. Identification of structures that could be contributing to the onset and progression of deformity will help medical professionals screen and treat potential problems that are not readily apparent to those with DMPN. Deformity might be prevented or minimized with an intensive program to strengthen the intrinsic foot muscles and limit stress on the posterior tibialis tendon (e.g. strengthening other plantarflexors, use of supportive orthoses). Despite encouraging findings, the results indicate that other factors, unaccounted for in this model, require further consideration. In particular, future work should develop measurement methods that would allow inclusion of ligament integrity.

Forty-four percent of midfoot function, measured as forefoot relative to hindfoot plantarflexion excursion during the heel rise task, was predicted by plantarflexor torque and intrinsic foot fat volume. It is interesting, but not surprising, that both, extrinsic and intrinsic muscles are important in midfoot function. The heel rise task requires a complex interaction between multi-joint plantarflexor force production and intact joint structure and function to transmit the forces that raise the body up onto the toes. The inability to plantarflex the forefoot on the hindfoot was weakly correlated with severity of midfoot deformity in those with DMPN (r=.19). Our data would suggest that loss of midfoot plantarflexion function is not unique to individuals with DMPN who have medial column deformity but that DMPN, in general, impairs plantarflexor and intrinsic muscles performance and results in poor midfoot function. It remains a reasonable hypothesis that impaired midfoot function transmits high deforming forces through the medial column of the foot. However given the prevalence of loss of midfoot function in those with DMPN, a longitudinal study will be required to understand the relationship between loss of midfoot plantarflexion function and deformity. Regardless of the role of movement on deformity development, it remains important to restore foot function and future research must examine whether the calf and foot muscles of individuals with DMPN are responsive to exercise and if midfoot function can be improved with a targeted exercise program.

When measures of muscle deterioration in DMPN are examined in comparison to the control group, those with DMPN had decreased muscle volumes and increased fat volumes in all muscles and compartments measured. The difference between groups in muscle and fat volumes was largest distally in the foot (intrinsic foot muscle=56% and intrinsic foot fat=67%) compared to proximally in the gastrocnemius (gastrocnemius muscle=31% and gastrocnemius fat=37%). This pattern of greater distal muscle deterioration compared to proximal muscle deterioration, is likely the result of the distal to proximal progression of neuropathy. In the proximal calf, however, it is interesting to note, similar to Tuttle et al[11], the greater and more consistent deterioration in muscle volume was observed in the gastrocnemius muscle. The gastrocnemius muscle appears to be at a uniquely high risk of deterioration. In addition, the strong positive correlation between gastrocnemius and deep compartment muscle volumes and plantarflexor torque links deterioration in muscle quality to deterioration in muscle function. Plantarflexor muscle function has long been associated with balance[40] and walking ability[41], both strongly associated with independent community function.[42]

Tendon volumes were not different between those with DMPN and controls. Our lack of difference in thickness between those with DM and controls mirrors a growing body of research which inconsistently finds thicker Achilles tendons in those with DM compared to controls.[17, 18, 38, 43-47] It is likely that changes in tendon size are impacted by more than presence or absence of DM. This is particularly true as we considered our *a priori* hypothesis regarding the posterior tibialis tendon and its relationship to deformity. If the posterior tibialis tendon were involved in deformity progression, we speculated that the tendon would thicken in the early phase of deformity and then thin to the point of rupture once deformity progression was severe.[48] There are a few points of data in the current sample, that suggest the presence of a non-linear pattern between tendon thickness as deformity severity, however there are too few study participants to fully examine this hypothesis.

There are 4 primary limitations associated with this study. First, the small sample size limited the number of potential predictors that could be examined related to midfoot alignment and function. Our current work focused on the relationship between muscle and tendon volumes and medial column deformity. Future work should also include measures of tissue quality as well as quantification of ligament integrity and joint congruity within the foot as they are also important in maintaining foot alignment. The small sample size also limited the ability to detect differences in muscle, fat, and tendon volumes in those with DMPN compared to controls and sample size calculations suggest group size would need to be tripled. The second limitation of this study was that the cross sectional design limits the ability to determine the cause/effect nature of the relationship. Progression of this original work looking at the relationship between key anatomical variables and deformity should include a longitudinal component of a large spectrum of individuals with DMPN. Third, there were measurement limitations. When measuring feet with deformity, whether by radiographs, MR, or kinematics, the measurements can be challenging as borders of tissues and palpable landmarks are not easily defined. Finally, the results of this study are limited to the predictors of alignment and function in individuals with diabetes of relatively long

duration (mean=17 years) and a high BMI. Future prospective studies should include those with low BMI and those with recently diagnosed DM to fully characterize the progression of foot deformity.

5. Conclusion

The deterioration of the supporting structures of the medial column (intrinsic foot muscle and fat volume, PT/FDL tendon volume, plantarflexor peak torque) predicts midfoot alignment and function. Prospective longitudinal research of individuals with minimal complications from diabetes should be conducted to monitor alignment, structure, and function over time to assist in developing early intervention strategies with the goal to prevent deformity, ulceration, and amputation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Feet of 23 participants with diabetes and peripheral neuropathy were studied
- Measures: radiographs, MRI, heel rise kinematics, and plantarflexor torque
- Predictors of medial column alignment: posterior tibialis tendon and foot muscle
- Predictors of medial column function: foot fat volume and plantarflexor torque
- Deterioration of medial column supports predicted alignment and function

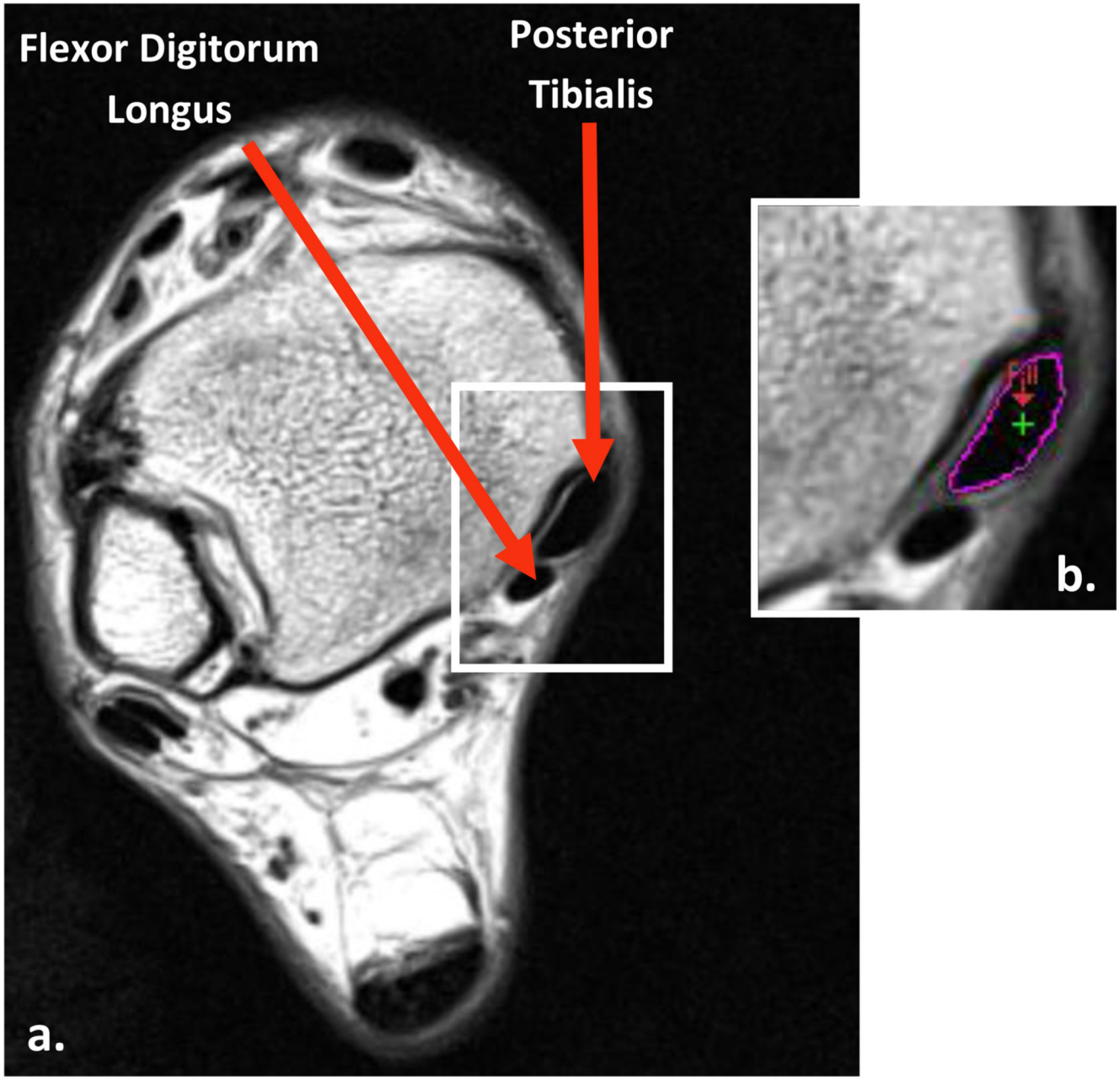

Figure 1.

a) Transverse magnetic resonance image of the distal leg. b) Image shows placement of the seed (+) and the tendon region that was grown (pink circle) by setting the intensity/ brightness threshold of the pixels that are included within the object boundary.

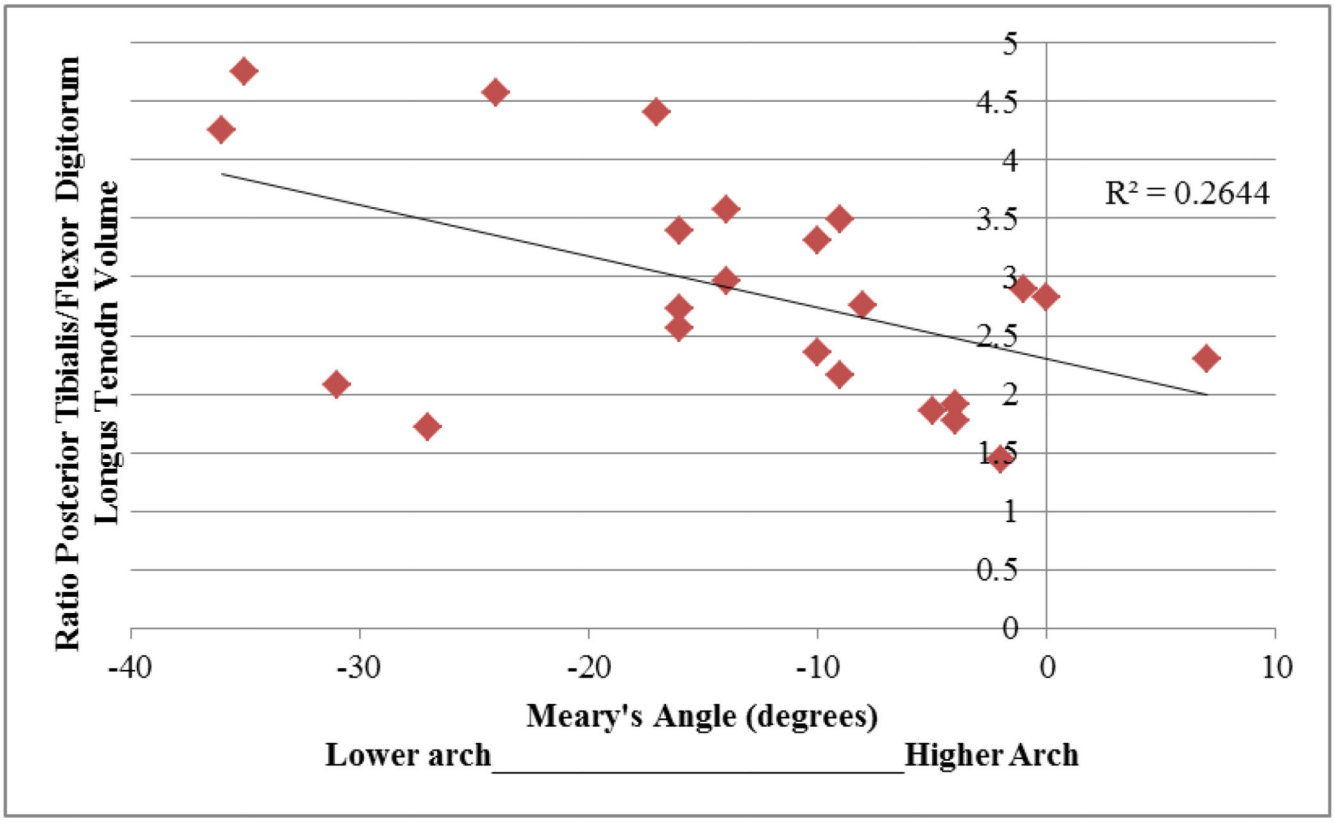

Figure 2.

Ratio of posterior tibialis to flexor digitorum longus tendon volume and Meary's angle in those with diabetes and peripheral neuropathy

There is a strong negative correlation between ratio of posterior tibialis to flexor digitorum longus volume and Meary's angle. As posterior tibialis tendon volume increases relative to the flexor digitorum longus volume, Meary's angle lowers (worsens). Also note that there are 2 individuals with severe deformity (large negative Meary's angle) who have a smaller posterior tibialis to flexor digitorum longus tendon volume ratio. This may indicate a non-linear relationship in which the posterior tibialis tendon thins or ruptures as deformity progresses.

Table 1

Subject Characteristics

	DMPN (n=23)	Controls (n=12)
Demographics		
Age (yr) *	59 (10)	57 (14)
Sex (Male/Female)	14/9	8/4
Height (m) *	1.73 (0.09)	1.74 (0.11)
Weight (kg) *	108 (27)	108 (30)
Body Mass Index (kg/m^2) *	36 (8)	35 (8)
Type of DM (1/2)	3/20	
Duration of DM (years) *	17 (11)	
Hemoglobin A1c (%) *	7.6^a (2.7)	5.2 (1.7)
Biothesiometry (volts) *	40^a (14)	20 (14)
Race (Caucasian/ African American)	17/6	8/4
Clinical Measures of Medial Column Alignment		
Calcaneal Eversion (degrees)	5 (5)	4 (4)
Medial Longitudinal Arch Angle (degrees)	137 (17)	145 (8)
Navicular Height (mm)	31^a (10)	38 (5)
Arch Index	0.316 (0.066)	0.344 (0.031)
Medial Midfoot Peak Plantar Pressure (kPa)	178 (227)	69 (65)

* Values are given as the mean (standard deviation) or number. Abbreviations: DMPN: diabetes mellitus and peripheral neuropathy

^{a} DMPN is different than Controls ($P<0.05$)

Table 2

Muscle, tendon, strength, fascia, and range of motion means (standard deviation). The correlation between the measures and Meary's angle and forefoot relative to hindfoot plantarflexion excursion (FF relative to HF excursion) are reported for the DMPN group.

	DMPN (n=23)	Control (n=12)	P value	Correlation to Meary's	Correlation to FF relative to HF excursion
Muscle Volume (cm^3)					
Gastrocnemius	110 (51)	150 (45)	.03	.20	.09
Soleus	114 (44)	142 (27)	.06	.23	−.18
Deep Posterior Compartment	49 (16)	62 (24)	.06	−.12	.12
Intrinsic Foot Compartment	18† (11)	32† (13)	<.01	.51*	.21
Fat Volume (cm^3)					
Gastrocnemius	42 (28)	29 (18)	.15	.41	−.22
Soleus	26 (20)	15 (5)	.07	.17	−.07
Deep Compartment	14 (9)	9 (4)	.08	−.12	−.26
Intrinsic Foot Compartment	18† (11)	9† (4)	.01	.08	−.35
Tendon and Fascia Volume (mm^3)					
Achilles	2638 (491)	2486 (679)	.48	.29	−.22
Posterior Tibialis	833 (227)	800 (220)	.69	−.28	−.03
Flexor Digitorum Longus	308 (90)	287 (71)	.54	.27	−.06
Flexor Hallucis Longus	337 (133)	341 (154)	.94	.16	−.08
Posterior Tibialis/Flexor Digitorum Longus Ratio	2.9 (1.0)	2.9 (0.7)	1.00	−.51*	.08
Torque (Nm)					
Plantarflexion	45.3[a] (15.1)	74.2 (33.0)	<.01	.21	.39
Heel Rise Kinematics (degrees)					
Forefoot Relative to Hindfoot Plantarflexion Excursion	3 (6)	13 (7)	<.01	.19	
Lateral Radiograph (degrees)					
Meary's Angle Toe Flat	−13 (11)	−11 5)	.48		.19
Meary's Angle Change (toe flat to toe extended)	8 (7)	10 (3)	.23	−.08	.33
Dorsiflexion (degrees)					

	DMPN (n=23)	Control (n=12)	P value	Correlation to Meary's	Correlation to FF relative to HF excursion
Prone with Knee Extended	2 (6)	3 (5)	.50	−.02	.17

†Data was previously published by Cheuy et al.[8]

Table 3

Multiple regression: Meary's angle (n=23)

	B	Standard Error B	β	p	Part Correlation
Constant	−5.67	8.43		0.51	
Posterior Tibialis to Flexor Digitorum Longus Tendon Volume Ratio	−5.10	2.15	−0.43	0.03	−0.42
Intrinsic Foot Muscle Volume	0.43	0.19	0.41	0.04	0.40
Plantar Fascia Function	−0.09	0.28	−0.06	0.75	−0.06
Dorsiflexion Knee Extended	0.00	0.35	0.00	1.00	0.00

R^2=0.44

Table 4

Multiple Regression: Forefoot relative to hindfoot plantarflexion excursion during heel rise (n=23)

	B	Standard Error B	β	p	Part Correlation
Constant	−0.77	4.57		0.87	
Intrinsic Fat Volume	−0.25	0.10	−0.47	0.03	−0.43
Posterior Tibialis Tendon Volume	−0.49	1.06	−0.09	0.65	−0.08
Plantarflexor Torque	0.26	0.09	0.52	0.01	0.49
Change in Meary's	0.19	0.14	0.24	0.20	0.24

R^2=0.44