Role of Intrinsic Muscle Atrophy in the Etiology of Claw Toe Deformity in Diabetic Neuropathy May Not Be as Straightforward as Widely Believed

SICCO A. BUS, PHD[1]
MARIO MAAS, MD, PHD[2]
ROBERT P.J. MICHELS, MD, PHD[1]
MARCEL LEVI, MD, PHD[1]

OBJECTIVE — Clawing of the toes in the diabetic neuropathic foot is believed to be caused by muscle imbalance resulting from intrinsic muscle atrophy. However, experimental data that support this mechanism are lacking. The aim of this study was to evaluate this hypothesis using magnetic resonance imaging (MRI).

RESEARCH DESIGN AND METHODS — In 20 neuropathic diabetic patients, 10 with claw toe deformity and 10 with normally aligned toes, multiple plane images of the foot and lower leg were acquired using T1-weighted spin-echo MRI. Atrophy of the intrinsic and extrinsic muscles controlling the toes was assessed using a semiquantitative 5-point atrophy scale. An intrinsic-to-extrinsic foot muscle imbalance score was derived from these atrophy scores, and correlation coefficients were established.

RESULTS — The mean ± SD intrinsic muscle atrophy score was 3.1 ± 1.1 for the toe deformity group and 2.6 ± 1.2 for the nondeformity group (not significantly different). The intrinsic muscle atrophy score was not significantly correlated with degree of toe deformity ($r = -0.18$). The muscle imbalance score was not significantly different between study groups and was not significantly correlated with degree of toe deformity ($r = -0.14$).

CONCLUSIONS — Neither intrinsic muscle atrophy nor muscle imbalance discriminated between neuropathic patients with or without claw toe deformity, suggesting that the role of these muscle factors in claw toe development may not be primary or as straightforward as previously believed. These findings shed new light on the etiology of foot deformity in diabetes and suggest a more complex nature of development, potentially involving anatomical and physiological predisposing factors.

Diabetes Care 32:1063–1067, 2009

Clawing or hammering of the toes is a common foot deformity in patients with diabetes, with reported prevalence values between 32 and 46% (1,2). Both claw and hammer toes involve hyperextension of the metatarsal-phalangeal (MTP) joint as the most important structural abnormality and will be referred to as "claw toes" in this article. Claw toes in diabetic patients are associated with a distal displacement of the protective submetatarsal head fat pads and an increase of plantar foot pressures at these regions (3,4). Furthermore, in prospective analyses, claw toe deformity has been found to be a predictor of diabetic foot ulceration (5), a condition that may lead to infection or amputation. Therefore, a proper understanding of the etiology of claw toe deformity is important if we are to initiate or improve interventions for the prevention or correction of claw toe deformity with which we can reduce the risk for ulceration and further complications in this patient group.

There are several theories that may explain why claw toes develop in diabetic patients. Ill-fitting footwear, in particular cramped toe boxes in patients who lack protective sensation, may externally force the toes in a clawed position (6). There are some suggestions that rupture of the plantar fascia or ligament contractures at the MTP joints may be involved (7,8). However, the most commonly reported cause of claw toe deformity is intrinsic muscle atrophy and weakness due to motor neuropathy, leading to an imbalance between intrinsic and extrinsic muscles across the MTP and interphalangeal joints (9–11). The long extrinsic flexors have a greater mechanical advantage over the extensors at the interphalangeal joints, and the extensors have a greater mechanical advantage over the flexors at the MTP joint (6,12). If the intrinsic muscles (i.e., the lumbricals and interossei) function correctly, they compensate for this mechanical advantage by flexing the MTP joint while extending the interphalangeal joints. But when the intrinsic muscles are atrophic and overpowered by the extrinsic muscles, this stabilizing action is lost, which eventually may result in clawing of the toes.

Despite the existence of numerous anecdotal and observational reports on this mechanical theory of claw toe pathogenesis and despite wide acceptance of this mechanism in the diabetic foot literature, experimental data to support this theory do not exist. In addition, some reservations regarding this mechanism were provided by results from recent studies. Bus et al. performed quantitative analyses of muscle atrophy and toe deformity using magnetic resonance imaging (MRI) and concluded that intrinsic muscle atrophy does not necessarily imply claw toe deformity in the diabetic neuropathic foot (13). In agreement with these results, Andersen et al. (14) found many of their neuropathic patients with normally aligned toes to have significant degrees of atrophy of the intrinsic foot muscles. Furthermore, van Schie et al. (15) found no

From the [1]Department of Internal Medicine, Academic Medical Center, University of Amsterdam, Amsterdam, the Netherlands; and the [2]Department of Radiology, Academic Medical Center, University of Amsterdam, Amsterdam, the Netherlands.
Corresponding author: Sicco A. Bus, s.a.bus@amc.uva.nl.
Received 5 December 2008 and accepted 2 March 2009.
Published ahead of print at http://care.diabetesjournals.org on 11 March 2009. DOI: 10.2337/dc08-2174.

association between semiquantitative scores of muscle weakness and foot deformity in diabetic male patients. Therefore, even though these findings do not suggest that intrinsic muscle atrophy and muscle imbalance are no longer contributing factors in the development of toe deformity, their role may not be as straightforward as widely believed.

A better understanding of the associations between muscle atrophy, imbalance, and toe deformity may be achieved with a more quantitative in vivo examination of both the foot and lower leg muscles using MRI in a group of patients with toe deformity and a matched group with normally aligned toes. Therefore, the purpose of this study was to use MRI to examine the extrinsic and intrinsic foot muscles in patients with claw toe deformity and matched patients with normally aligned toes to explore in a more objective manner the association between intrinsic muscle atrophy and claw toe deformity. Based on the current widespread beliefs, we intended to test the hypothesis that clear differences in both intrinsic muscle atrophy and muscle imbalance are present between patients with toe deformity and those without.

Table 1—*Baseline patient characteristics and study results*

	Experimental group: claw toe deformity	Control group: aligned toes
n	10	10
Sex (male/female)	6/4	6/4
Age (years)	58.5 ± 7.1	58.7 ± 6.2
Height (m)	1.74 ± 0.08	1.73 ± 0.05
Weight (kg)	83.3 ± 14.2	81.6 ± 10.2
BMI (kg/m^2)	27.3 ± 3.2	27.4 ± 3.8
Diabetes type (1/2)	6/4	8/2
Diabetes duration (years)	34.6 ± 11.9	31.3 ± 14.6
A1C (%)	7.4 ± 1.0	8.0 ± 1.0
Neuropathy duration (years)*	12.7 ± 5.5	11.9 ± 8.5
Vibration perception threshold (V)	32.6 ± 13.9	37.7 ± 10.3
Toe angle (°)	−21.0 ± 5.6	−3.1 ± 7.0†
Intrinsic muscle atrophy score	3.1 ± 1.1	2.6 ± 1.2
EDL atrophy score (proximal)	0.5 ± 0.5	0.4 ± 0.7
EDL atrophy score (distal)	1.1 ± 1.1	0.8 ± 1.3
FDL atrophy score (proximal)	0.1 ± 0.3	0.2 ± 0.6
FDL atrophy score (distal)	0.2 ± 0.4	0.7 ± 1.1
Intrinsic-to-extrinsic muscle imbalance score	2.2 ± 1.1	2.0 ± 1.0
Extrinsic muscle fibrosis	0	0

Data are means ± SD or *n*. *As derived from medical records or, when absent, estimated by the patient based on the first appearance of neuropathic symptoms. †$P < 0.001$ between groups.

RESEARCH DESIGN AND METHODS

— Twenty diabetic patients with distal symmetric polyneuropathy participated in this cross-sectional study. Ten of these patients (five men and five women) had claw toe deformity involving hyperextension of the MTP joint (experimental group). The other 10 patients were matched on age (±5 years) and sex and had normally aligned toes (control group). Five age-matched healthy subjects (three men and two women) with normally aligned toes were included for reference purposes in MRI assessments. The presence of toe deformity was initially assessed clinically for recruitment purposes but eventually based on MRI analysis as described below. One lower extremity per subject was examined because of the limited time available per patient on the MRI scanner. This was the extremity with toe deformity if the toes of the contralateral foot were not deformed or was randomly assigned if not excluded by the criteria mentioned below.

Distal symmetric polyneuropathy was assessed clinically and confirmed to be present in all patients by abnormal vibration perception thresholds measured at the dorsal surface of the hallux in both feet using a Biothesiometer (Bio-Medical Instrument Company, Newbury, OH) (16), and the inability to sense the pressure of a 10-g (5.07) Semmes-Weinstein monofilament at, at least, one of eight sites tested (six plantar foot regions, dorsum of the foot, and medial malleolus). Written informed consent was obtained from each subject before the start of the study, which was approved by the local medical ethics committee. Patient characteristics are summarized in Table 1.

Maximal effort was made to exclude congenital or external causes of claw toe deformity in the experimental group. For this purpose, the patients' shoes were examined, and patients were asked about the onset of their deformity and the fitting of their shoes in the past. Patients were excluded if their shoes were found to be too small in size for their feet, if they reported having worn ill-fitting shoes in the past, or if toe deformity was present before the onset of diabetes. For the same reason, patients with neuromuscular diseases or neurological problems other than diabetic polyneuropathy were excluded. Other exclusion criteria were *1*) age <40 or >65 years; *2*) peripheral vascular disease, as determined by absent pedal pulses with an ankle-brachial index <0.75 or toe pressure <50 mmHg; *3*) a current foot ulcer, a prior ulcer at the metatarsal heads, or prior lower-extremity surgery or fracture; *4*) rheumatoid arthritis, lower-extremity amputation, or Charcot neuro-osteoarthropathy; and *5*) conditions precluding MRI assessment. None of the five healthy nondiabetic subjects had any known (history of) foot pathological conditions.

Procedures

A 1.5-T Magnetom Vision imager (Siemens, Erlangen, Germany) was used to acquire T1-weighted spin-echo series of the foot and lower leg. The subject lay supine with the foot or leg inserted into a circular polarized head coil (17). In a comfortable position at ~30° plantar flexion, the foot was immobilized using padding material without affecting the natural configuration of the toes. The foot was imaged in a sagittal and coronal (axial) plane view, and the lower leg was imaged in a transverse (axial) plane view. Two separate datasets, distal and proximal, were acquired for the lower leg because of the limited field of view (FOV) of the coil used. For all images collected, repetition time was 577 ms, echo time was 17 ms, and slice thickness was 3 mm. The sagittal plane dataset of the foot was oriented parallel to the second metatarsal bone and consisted of 19 slices acquired between the first metatarsal head medially and the fifth metatarsal head laterally with

FOV 256 × 256 mm, in-plane resolution 512 × 512 pixels, and interslice gap 0.9 mm. The coronal plane dataset of the foot was oriented perpendicular to the sagittal plane images and consisted of 20 slices collected between the proximal phalanges distally and the cuneiform bones proximally with FOV 150 × 150 mm, resolution 256 × 256 pixels, and 0.9 mm interslice gap. The lower leg datasets were oriented perpendicular to the long axis of the tibial bone in a coronal and sagittal plane view and each consisted of 20 slices with FOV 200 × 200 mm, resolution 256 × 256 pixels, and interslice gap 5 mm. The distal lower leg dataset included the ankle joint, and the proximal dataset included the knee joint. Total data acquisition time was 45 min per subject.

Toe deformity was assessed non–weight bearing from the sagittal plane images using IMPAX WEB1000 software (Agfa-Gevaert, Mortsel, Belgium) by measuring the angle between a line parallel to the sole of the forefoot and the bisector of the proximal phalanx of the second or third toe (named "toe angle," with negative values denoting extension). Toe angles smaller than −13° indicated deformity based on 95% normal limits (3).

Atrophy of the intrinsic muscles in the forefoot (i.e., the interossei and lumbricals) was assessed from the coronal plane foot images. On these images, muscle is represented by a low-intensity (dark gray) signal, whereas fatty infiltration (atrophy) of the muscle shows as a high-intensity (light gray) signal. Because atrophy was diffusely distributed throughout the muscle, one representative anatomically referenced image cutting through the fifth metatarsal head was selected to score degree of intrinsic muscle atrophy. For this purpose, we used a semiquantitative 5-point atrophy scale with 0 representing healthy muscle tissue (no atrophy), 1 representing mild atrophy, 2 representing moderate atrophy, 3 representing severe atrophy, and 4 representing almost complete or complete loss of muscle tissue. Reliability in assessing atrophy using this 5-point scale was high, with weighted κ of 0.94 (18).

Extrinsic foot muscle status was assessed using both sets of lower leg images. The extensor digitorum longus (EDL) and flexor digitorum longus (FDL) muscles were evaluated using all proximal to distal images from the knee to the ankle on which these muscles could be identified (Fig. 1*A* and *B*). Extrinsic foot muscle atrophy was scored using a semiquantitative 5-point atrophy scale similar to that used for the intrinsic foot muscles. Proximal and distal portions of the muscle were scored separately (division at mid-tibia). A muscle imbalance score between the intrinsic and extrinsic muscles, which act at the level of the MTP joint, was defined by subtracting the intrinsic muscle atrophy score with the average proximal and distal EDL muscle atrophy score. The possible range of muscle imbalance scores was between 0 and 4, with 0 representing no muscle imbalance and 4 representing maximal muscle imbalance. Only the atrophy scores of the EDL muscle were entered in the equation because, according to the theory, this muscle acts in overpowering the intrinsic muscles at the MTP joint, which is the primary joint in claw toe deformity. The presence of intramuscular fibrosis indicating muscle contractures, which may also play a role in overpowering the intrinsic muscles (19,20), was scored as a hypointense (black) signal on the transverse plane images of the lower leg (21).

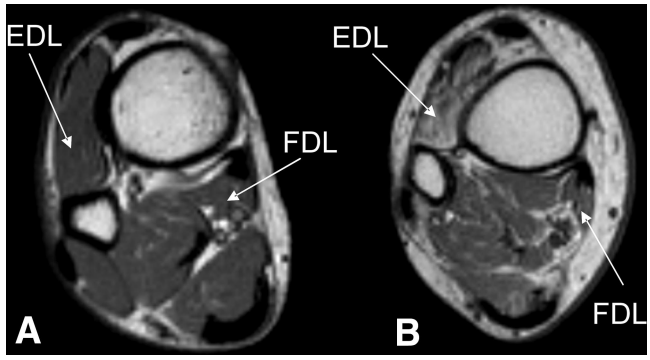

Figure 1—*Cross-sectional images of the distal lower leg in a healthy nondiabetic subject* (A) *and a neuropathic patient with severe atrophy of the EDL and mild atrophy of the FDL* (B). *The EDL and FDL muscles were scored proximally and distally by sequentially examining all cross-sectional images that included these extrinsic foot muscles.*

Two observers, blinded for patient identity and study group, independently performed assessments of muscle atrophy. They reached a consensus regarding outcome when discrepancies in observations were found.

Statistical methods

SPSS (version 16.0) was used for statistical analysis. For all continuous data, independent *t* tests (normally distributed data) and Mann-Whitney nonparametric tests (skewed data) were used to compare study groups. Fisher's exact test was used to compare groups for dichotomous data. Spearman rank correlation coefficients were computed for associations between selected variables in the pooled group of patients ($n = 20$). For all tests, significance levels of $P < 0.05$ were used.

RESULTS — Except for toe angle, no significant differences were present between subject groups for baseline data (Table 1). Some degree of intrinsic muscle atrophy (minimum score 1) was present in each of the 20 neuropathic feet examined, and the whole range of atrophy scores (1–4) was represented in both groups. Twelve patients, seven in the experimental group and five in the control group had severe or worse degrees of atrophy (score 3 or 4). The mean ± SD atrophy scores were 3.1 ± 1.1 for the experimental group and 2.6 ± 1.2 for the control group, which were not significantly different ($P = 0.34$) (Table 1). The correlation coefficient between the intrinsic muscle atrophy score and toe angle was −0.18 (not significant, $P = 0.44$). Figure 2 shows two examples: one of an experimental group patient with a severe deformity (toe angle −26.3°) but with only a mild degree of intrinsic muscle atrophy (score 1) and a control group patient with almost no intrinsic muscle left (score 4) but with normally aligned toes. None of the five healthy nondiabetic subjects showed any degree of intrinsic muscle atrophy (all subjects had score 0).

Neither the FDL nor the EDL muscle showed any hypointense signal indicating fibrosis on the lower leg MRI scans. The EDL muscle was atrophic in six experimental and four control group subjects with more atrophy present distally (Table 1). The FDL muscle was atrophic in three experimental and four control group patients. In those experimental group patients showing FDL muscle atrophy, a score >1 (i.e., mild atrophy) was not found. The EDL muscle was slightly more

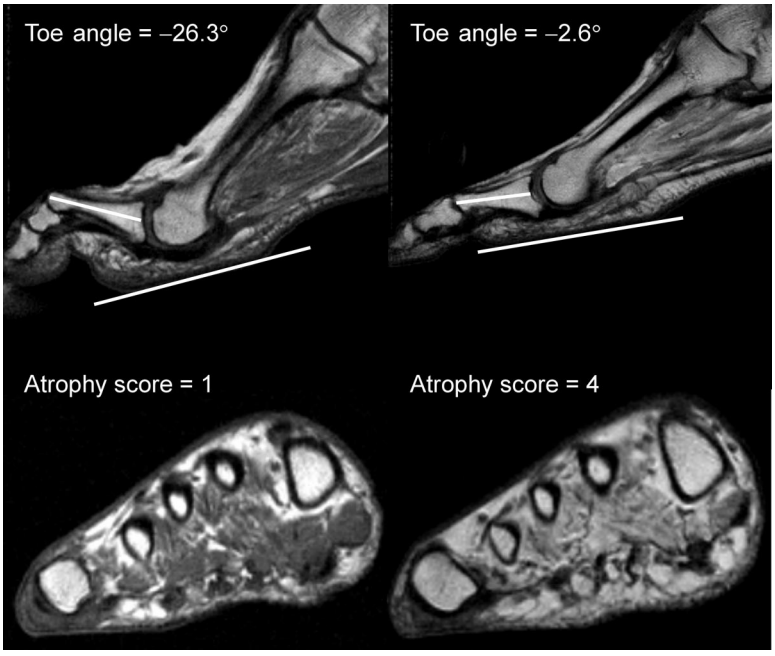

Figure 2—*Two cases illustrating the lack of association between intrinsic muscle atrophy and claw toe deformity. Sagittal and coronal plane foot images of a patient with severe deformity but only mild atrophy* (left panel) *and a patient with perfectly aligned toes but almost no intrinsic muscle left in the foot* (right panel).

atrophic than the FDL muscle. No significant differences were found between experimental and control groups in any of the extrinsic muscle atrophy scores. The intrinsic muscles were more atrophic than the extrinsic muscles, with muscle imbalance scores of 2.2 and 2.0 for the experimental group and control group, respectively. The difference in muscle imbalance score between the two groups was not significant ($P = 0.60$). The correlation coefficient between muscle imbalance score and toe angle was −0.14 (not significant, $P = 0.56$).

CONCLUSIONS — The results of this study showed no significant difference in degree of intrinsic muscle atrophy between matched patients with claw toe deformity and patients with normally aligned toes. Furthermore, no association was found between intrinsic muscle atrophy score and degree of deformity (toe angle) in the group of 20 patients tested. This finding is clearly illustrated by the two cases shown in Fig. 2. In the extrinsic foot muscles, we found no signs of fibrosis in either group that may indicate the presence of muscle contractures nor did we find significant differences in extrinsic muscle atrophy scores between the groups. Furthermore, the intrinsic-to-extrinsic muscle imbalance score was not different between groups and not associated with toe angle. This result means that neither intrinsic muscle atrophy nor muscle imbalance was able to discriminate between neuropathic patients with claw toe deformity and those without. To the best of our knowledge, this is the first study that measures intrinsic-to-extrinsic foot muscle imbalance in the diabetic foot and attempts to associate intrinsic muscle atrophy and muscle imbalance with claw toe deformity in an objective manner.

The present data confirm previous MRI reports on intrinsic muscle status in diabetes from Bus et al. (13) and Andersen et al. (14), who also showed substantial degrees of intrinsic muscle atrophy in neuropathic diabetic feet. Regarding the association between muscle status and foot deformity, our findings are in agreement with data from van Schie et al. (15), who showed that muscle weakness in the foot, assessed using manual muscle testing, was not associated with foot deformity in diabetic patients. Based on the lack of associations found between muscle atrophy or muscle imbalance and claw toe deformity, the present results suggest that the widely reported theory that intrinsic muscle atrophy and loss of muscle balance cause claw toes in the diabetic foot should be treated with caution. This does not mean, however, that we suggest that muscle atrophy and imbalance are no longer permissive factors in claw toe etiology. All patients with toe deformity in our study had at least some degree of intrinsic muscle atrophy (score ≥1), and because intrinsic muscle atrophy can precede toe deformity (14), it may still be a contributing factor. Nevertheless, this relationship may not be straightforward in the diabetic foot. Other factors may be (more) important with the likelihood that multiple factors are present to explain the presence of claw toe deformity in diabetic patients.

One of these factors may be pathology of the plantar aponeurosis, an important connective tissue structure that contributes to MTP joint stability by providing plantar flexion at this joint during weight-bearing. On MRI, Taylor et al. (8) consistently found plantar aponeurosis discontinuity, indicating rupture, in diabetic patients with claw toes and normal aponeurosis appearance in patients with aligned toes. With rupture, the aponeurosis would lose its stabilizing properties, which may draw the MTP joint in a hyperextended position. However, robust studies that support these preliminary data have not been found. Another factor may be pathological changes of the MTP joint capsule, including the plantar plate and collateral ligaments. Some authors studying nondiabetic subjects have associated rupture or degeneration in these structures with MTP joint instability and toe deformity (19,22). In diabetes, these soft tissue abnormalities in the foot may be fueled by the process of nonenzymatic glycosylation, which renders fascia and ligaments less functional in their capacity to control joint configuration in the forefoot. Finally, externally applied forces through the effect of ill-fitting footwear may play a role, in particular when the intrinsic foot muscles are no longer able to contribute to MTP and interphalangeal joint stabilization due to atrophy and weakness. Clearly, prospective studies are needed to show the validity of these alternative mechanisms in contributing to claw toe deformity in the diabetic patient.

Our findings may have implications if surgical corrective interventions are the choice of treatment for claw toes in diabetic patients. Extensor tenotomies may not have the desired outcome and a multitissue approach may be warranted, an observation that is supported by the work in nondiabetic feet from Myerson and Shereff (7), who suggested that the correction of claw toes may require more ex-

tensive sectioning than formerly believed. This includes sectioning of the MTP joint collateral ligaments. Whether this also applies to the diabetic neuropathic foot remains to be investigated.

This study was limited in that the cross-sectional design did not allow the establishment of cause-and-effect relationships. Long-term follow-up of patients with variable degrees of toe deformity may further improve our understanding of its pathogenesis. Second, we did not assess the physiological effects of diabetes and neuropathy on muscle tissue properties, which may influence muscle function and the associations sought between foot structure parameters. ^{31}P magnetic resonance spectroscopy can be used for this purpose, including the measurement of nonenzymatic glycosylation and metabolite concentrations (23,24) and may be found valuable in future studies on claw toe pathogenesis. Finally, this study should be considered as a first attempt to obtain objective data on the association between muscle atrophy, imbalance, and toe deformity with the goal of increasing awareness for the relationship between these parameters that is possibly more complex than previously thought. We expected to find clear and consistent differences between the two study groups for the muscle factors evaluated, which, if present, would indicate their potential importance in the etiology of claw toe deformity.

In summary, our results suggest that the role of intrinsic muscle atrophy and muscle imbalance in explaining the presence of claw toe deformity in the diabetic foot may not be as straightforward as widely believed. These muscle factors may not be primary or solely responsible for the development of claw toe deformity in diabetes. Other (predisposing) internal or external factors may be (more) important contributors, either in causing toe deformity or in preventing the establishment of a clear relationship between muscle atrophy and toe deformity. All of these factors may also act together in a multicomponent fashion. Prospective studies are required to test these hypotheses. It is hoped that the findings from this study will provoke further research that eventually may lead to the acceptance or rejection of the "muscle atrophy and imbalance" theory in the development of claw toe deformity in the diabetic foot.

Acknowledgments— No potential conflicts of interest relevant to this article were reported.

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