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Clawed toes in the diabetic foot: neuropathy, intrinsic muscle volume, and plantar aponeurosis thickness

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Introduction

Clawed toes, defined as extension of the metatarsophalangeal joint (MTPJ) and flexion of the proximal and distal interphalangeal joints (IPJ), have been associated with the diabetic foot. One theory states that this deformity is caused by an imbalance between the extrinsic and intrinsic foot muscles [1,2]. However, Bus *et al.* found a 73% decrease in intrinsic muscle cross sectional area between diabetic neuropathic patients and controls, but only 2 of 8 neuropathic patients had toe deformities [3]. Anderson *et al.* found that diabetic neuropathic patients had a little more than 50% of the intrinsic muscle volume of either controls or non-neuropathic diabetic patients, but none of the diabetic neuropathic patients had toe deformities [4]. Others have found a link between plantar apo-neurosis (PA) dysfunction and clawed toes [5,6] and between diabetes and a thicker PA [7-9]. The purpose of this study was to explore the relationship between claw toes, neuropathy, intrinsic muscle volume and PA thickness.

Methods

We enrolled 40 diabetic subjects in 4 groups: G1) neuropathic, claw toes, G2) neuropathic, no claw toes, G3) non-neuropathic, claw toes, and G4) non-neuropathic, no claw toes. We have analyzed a subset for this abstract (G1: n = 6, G2: n = 4, G3: n = 6, G4: n = 4). The presence of claw toes was determined via clinical exam and neuropathy was defined as insensitivity to a 10 g monofilament. Partial weight-bearing CT scans were taken for each foot. Intrinsic muscle volume was determined by segmenting

each foot using MultiRigid; data were normalized to total foot volume. PA thickness was measured at 1/5 the distance from the heel to the base of the first metatarsal using ImageJ. A two-way analysis of variance was used to test for interaction and significance ($p < 0.05$).

Results

There was significant interaction between foot deformity and neuropathy ($p = 0.02$). Feet with both neuropathy and foot deformity had lower mean volume than all the other groups, i.e., volume was reduced only when feet were both neuropathic and had deformity (Table 1).

Mean PA thickness was significantly higher for feet with foot deformities than those without ($p = 0.019$) (Table 2). Thickness was also higher in neuropathic feet than in non-neuropathic feet, but this difference was not significant ($p = 0.14$). While the data suggests that thickness was highest in feet that had both neuropathy and deformity beyond the additive effects of each factor separately, the interaction between deformity and neuropathy was not significant ($p = 0.2$).

Table 1: Intrinsic muscle volume (mean \pm SD)

	claw toes yes	claw toes no
neuropathy yes	0.130 \pm 0.037	0.191 \pm 0.008
neuropathy no	0.198 \pm 0.018	0.192 \pm 0.034

Table 2: PA thickness (mean \pm SD)

	claw toes yes	claw toes no
neuropathy yes	4.57 \pm 1.19	2.96 \pm 0.22
neuropathy no	3.51 \pm 0.99	3.00 \pm 0.20

Conclusion

Our pilot study demonstrates that neuropathic feet with claw toes have less intrinsic muscle volume than the other groups. The same feet also had thicker PA, suggesting that both intrinsic muscle atrophy and PA dysfunction are required for the development of claw toes. The specific mechanism of clawing with a thicker PA (as opposed to a ruptured PA, as seen previously [5,6]) is not yet understood.

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