ALTERED FOOT LOADING IN DIABETICS. THE ROLE OF ACHILLES TENDON AND PLANTAR FASCIA.

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INTRODUCTION

The diabetic foot often undergoes abnormal plantar pressures, changing in walking strategy, ulcerative processes. Neuropathy was frequently added as the main responsible for the alteration of foot loading pattern [1]. A different approach comes from the observation that the common sign of the distinct diabetic syndromes, hyperglycaemia, promotes glycosilation of proteins and the consequent accumulation of advanced end-products in most human tissues [2]. This means that muscles, cartilages, tendons, ligaments, all might experience structural changes even before the onset of diabetic neuropathy, and might then concur to alter the gait pattern.
The present study focuses on the effects that diabetes-induced alterations of Achilles tendon, plantar fascia and 1st metatarso-phalangeal joint – both anatomical and functional - may have on foot loading.

METHODS

Sixty-one diabetic patients (27 without neuropathy, 19 with neuropathy, 15 with previous neuropathic ulcers), and 21 healthy volunteers were recruited. Thickness of Achilles tendon and plantar fascia was measured by ultrasound [3]. Flexion-extension of the 1st metatarso-phalangeal joint was measured passively by a long-arm mechanical goniometer [3]. Main biomechanic parameters of foot-floor interaction during gait were acquired by means of an integrated force/pressure device [3]. Among them, only vertical ground reaction (expressed as a percentage of body weight (b.w.)), force/time integral and relative loading time (expressed as a percentage of the whole stance phase) under the metatarsals were included in the present study. Piecewise linear regressions were applied to relate all the above measurements.

RESULTS AND DISCUSSION

Plantar fascia and Achilles tendon were significantly thicker in diabetics with neuropathy than in controls, while flexion-extension of the 1st metatarso-phalangeal joint was significantly smaller. Load under the metatarsal heads significantly increased in diabetics with neuropathy in terms of both amplitude and duration, as proved by vertical forces and integrals data. Mean values and standard deviations of all the above parameters are reported in Table 1. The increase in the vertical force under the metatarsals strongly related (R=0.83, explained variance = 70.1%) with the changes in the three examined structural and functional parameters.
Thickening of plantar fascia and Achilles tendon in diabetics, more evident in the presence of neuropathy, concurs to develop a rigid foot, which poorly performs the physiological impact force absorption during landing. More generally, an overall alteration of the foot-ankle complex motion likely occurs throughout the whole gait cycle, which partly explains the abnormal loading under the forefoot.

CONCLUSIONS

Even though neuropathy is still considered as the major responsible for the ulceration of the diabetic foot, we believe that several other factors should be investigated and monitored, in the attempt of preventing the formation of such severe wounds.
In the present study we observed structural alterations of the main tendinous and ligamentous structures of the foot-ankle complex in presence of mild-to-severe diabetic neuropathy. Range of motion of the 1st metatarso-phalangeal joint in the sagittal plane was also measured under passive conditions. Most important finding was that the thicker the Achilles tendon and plantar fascia become, and the more the 1st metatarso-phalangeal joint mobility is reduced, the more severe is the overall alteration of the foot loading during gait. More specifically, concurrent changes of the above factors accounted for 70.1% of the changes in metatarsals loading for all diabetics groups, with and without neuropathy. In presence of severe neuropathy only, the explained variance attained a value of 74.4%.
We do not claim the hereby measured alterations as the only factors which are responsible for the triggering of the neuropathic ulceration process. Further studies are needed to merge the contributions of the numerous structural and functional alterations of the diabetic foot, which concurrently take the forefoot plantar soft tissue under conditions of "potential damage" [4].

REFERENCES

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Table 1: Mean values and standard deviations of the measured parameters for Healthy Volunteers (HV), Diabetics without neuropathy (D), Diabetics with Neuropathy (DN), Diabetics with Previous Neuropathic Ulcers (DPNU). Statistically significant differences (*P<0.05) were assessed by means of one-way ANOVA and Bonferroni *post hoc* test.

	Achilles tendon thickness (mm)	Plantar fascia thickness (mm)	1st met-phal joint flexion-extension (°)	Metatarsals vertical forces (% b.w.)	Metatarsals integrals (%b.w.*ms)	Metatarsals loading time (%stance)
HV	4.0 ± 0.5	2.0 ± 0.5	100.0 ± 10.0	89.9 ± 6.3	2956.8 ± 430.5	88.2 ± 4.0
D	4.6 ± 1.0	2.9 ± 1.2	54.0 ± 29.4	93.9 ± 6.7	3209.7 ± 493.3	90.1 ± 4.7
DN	4.9 ± 1.7*	3.0 ± 0.8*	54.9 ± 17.2*	96.0 ± 7.0*	3625.2 ± 695.3*	91.7 ± 6.6
DPNU	5.2 ± 1.7*	3.1 ± 1.0*	46.8 ± 20.7*	97.5 ± 7.0*	3722.1 ± 743.2*	93.0 ± 6.3*