Reduced lower limb muscle strength and volume in patients with Type 2 diabetes in relation to neuropathy, intramuscular fat and vitamin D levels

Monirah M. Almurdhi, MSc[1], Neil D. Reeves, PhD[2], Frank L. Bowling, DPM, PhD[1], Andrew J.M. Boulton, MD, FRCP[1], Maria Jeziorska, MD, PhD[1], Rayaz A. Malik, MBChB, PhD[1,3]

Centre for Endocrinology and Diabetes, Institute of Human Development, University of Manchester and Central Manchester NHS Foundation Trust, Manchester Academic Health Science Centre, UK[1], School of Healthcare Science, Faculty of Science and Engineering, Manchester Metropolitan University, Manchester, United Kingdom[2], Weill-Cornell Medical College, Doha, Qatar[3]

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Address of correspondence and reprint requests to:
Professor Rayaz A. Malik
Professor of Medicine and Consultant Physician
Weill Cornell Medical College, Qatar & University of Manchester School of Medicine
Centre for Endocrinology and Diabetes, Institute of Human Development
46 Grafton Street, Core Technology Facility
Manchester, M13 9NT
T: 0161 275 1196, F: 0161 275 1183
E: ram2045@qatar-med.cornell.edu & rayaz.a.malik@manchester.ac.uk

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We thank Treiber et al for sharing their data in relation to a greater prevalence of painful neuropathy and slower walking speeds in their patients taking a statin [1]. They suggest that some of the differences observed in our study could be explained by treatment with statins. We have utilised our data to assess if statins may contribute to lower limb weakness or muscle atrophy and neuropathy.

We have compared our study parameters between T2DM patients on a statin (n=16) to those who were not on a statin (n=4; Table 1). The interpretation of our data is limited and cautious because of the small number of patients not on a statin and also the lack of matching for age, weight and BMI. Nevertheless, muscle strength of the lower limb (knee extensors and ankle plantar flexors) did not differ significantly. Muscle volumes for the knee extensors (P=0.04) and flexors (P=0.09) were lower, with no difference in the ankle plantar (P=0.21) and dorsiflexor (P=0.24) muscle volumes of T2DM patients on a statin compared to patients not on a statin.

Diabetic patients on a statin were older and it is well known that muscle mass declines as a result of ageing due to a reduction in skeletal muscle fibre number, size and length [2]. Whilst it has been suggested that a reduction in muscle size can result in reduced motor neuron unit activation and decreased muscle force and power generation [2], this was not observed in the present study. Another possible explanation is that the older individuals on a statin have a reduction in physical activity, which particularly affects the antigravity muscles such as the knee extensors. Reduced muscle size, reduced muscle activation capacity and ageing are of course highly correlated with reduced muscle strength [3], but in the present study there was no difference between those patient taking a statin compared to those not on a statin.

The loss of muscle mass is also associated with diabetic neuropathy, and in the present study vibration perception threshold (VPT) (P=0.0001) and NDS (P=0.09) were significantly higher indicative of neuropathy in diabetic patients on a statin compared to patients not on a statin, similar to the findings of Treiber et al. Of course whether or not a T2DM patient is on a statin will always be confounded by age, cardiovascular risk and the presence of other microvascular complications [4]. Furthermore, contrary to the studies cited by Trieber and colleagues, a recent large study has shown that treatment with statins may prevent the development of diabetic neuropathy [5]. And of course whether or not a patient is taking a statin depends on whether they can tolerate it, particularly in relation to vitamin D deficiency [6]. A large prospective study is required to establish the potential relationship between statin use, muscle volume and strength and indeed walking ability and falls in diabetes.

References

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Table 1. Clinical demographics and measures of neuropathy, muscle strength and volume in T2DM patients treated with or without a statin.

Variables	T2DM-No Statin (n=4/20)	T2DM- Statin (n=16/20)	P value
Age (years)	50±13.4	66±7.5	0.09
Body mass (kg)	97.6±18.8	79.1±16.7	0.14
BMI (kg/m^2)	32.7±5.6	28.5±3.4	0.24
VPT (Hz)	**5.3±1.3**	**15.7±8.4**	**0.0001**
NDS (0-10)	1.2±1.8	3.5±2.5	0.09
Muscle strength (Nm/kg)			
Knee extensors	1.4±0.4	1.3±0.5	0.47
Ankle plantar flexors	0.8±0.3	0.6±0.2	0.28
Muscle volume (cm^3)			
Knee extensors	**1370.9±316.1**	**873.7±356.2**	**0.04**
Knee flexors	705.1±199.3	473.4±204.8	0.09
Ankle plantar flexors	882.0±323.3.7	629.9±155.2	0.21
Ankle dorsiflexors	253.9±83.9	192.4±39.6	0.24

Data are expressed as Mean ± SD. BMI: Body mass index; VPT: Vibration perception threshold; NDS: Neuropathy disability score.