

SUB-CALCANEAL FAT-PAD INFILTRATION AND ITS EFFECT ON PLANTAR HEEL PRESSURES IN THE DIABETIC NEUROPATHIC FOOT

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INTRODUCTION

Diabetes mellitus and its complications are associated with several structural and physiological abnormalities in the foot, which can increase the risk for plantar ulceration [1]. These abnormalities also include changes in the sub-calcaneal and sub-metatarsal fat-pads used for cushioning. Micro-hemorrhage of sub-cutaneous tissue has been reported [1,2] and non-enzymatic glycosylation of proteins may lead to systemic fat pad tissue changes [3]. The purpose of this study was to use magnetic resonance imaging (MRI) to quantify pathologic changes in the sub-calcaneal fat pads and to determine the association with plantar pressures measured in the heel region in diabetic patients with peripheral neuropathy.

METHODS

Fourteen diabetic patients with peripheral neuropathy (mean age 57.9 years (SD 6.9), body mass 81.7 kg (SD 11.4)) and five healthy age-matched control subjects (mean age 58.0 years (SD 1.7), body mass 73.4 kg (SD 6.1)) underwent MRI examination and plantar foot pressure measurement. Two-point Dixon chemical shift imaging was performed at 1.5-T using a Siemens Magnetom 63SP/4000 imager. Fat-only and water-only (fat-suppressed) images were created from which the fraction of fat signal in each pixel could be determined [4]. All images were high-resolution (512x512 pixels) T₁-weighted sagittal plane spin-echo images of the foot and were obtained non-weight bearing. One centrally located slice was selected for quantitative analysis. Using Scilimage a region of interest (ROI) was defined in the heel and the fat signal fraction in this ROI was calculated (Figure 1).



Figure 1: Water-only image in which ROI was defined between 1/12 and 3/12 of the foot length from the heel

Barefoot plantar pressures during gait were measured using an EMED pressure platform (Novel, Germany) while obtaining a second-step approach to the platform. Five repeated trials were collected from each subject. Peak pressures were calculated for the heel region. Mann-Whitney non-parametric tests were used to determine statistical significance between the groups ($P < 0.05$).

RESULTS

The mean fat signal fraction was 0.72 (SD 0.03, range 0.70-0.76) for the healthy control subjects and 0.55 (SD 0.11, range 0.34-0.67) for the diabetic patients; the mean difference was statistically significant ($P < 0.005$). Peak pressure in the heel was 325 kPa (SD 53) and 391 kPa (SD 119) for the healthy controls and diabetic patients, respectively, which was not significantly different. Figure 2 shows examples of healthy and infiltrated sub-calcaneal fat-pad tissue. A significant inverse correlation of -0.59 was present between fat signal fraction and heel peak pressure ($P < 0.01$).

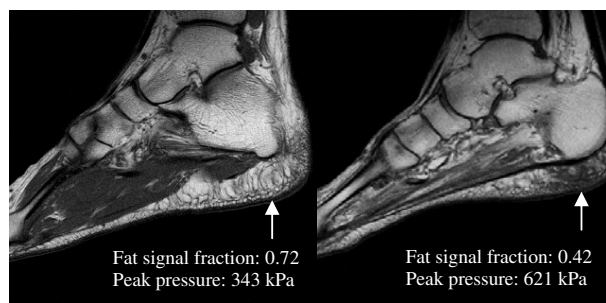


Figure 2: Healthy subject with normal sub-calcaneal fat pads (left) and diabetic patient showing fat-pad infiltration (right)

DISCUSSION

A substantial reduction in the ratio of fat to water signal in the sub-calcaneal fat pads was shown in a large percentage of neuropathic patients when compared with healthy control subjects. These results are consistent with data from Kao et al. [3] showing increased T₁-relaxation times in cadaver diabetic heel fat pads. Increased amounts of collagen as a result of non-enzymatic glycosylation or micro-hemorrhage caused by repetitive minor trauma may explain these pathologic changes [1,2,3]. The data suggests reduced functionality of the sub-calcaneal fat pad as shown by the significant association between reduced fat signal fraction and increased pressure levels in the heel. Whether fat-pad infiltration or elevated mechanical pressure is the primary event in this association requires further prospective analysis. The findings from this study contribute to our understanding of the relationship between structural and functional parameters in the diabetic foot. Although heel ulcers are not common in the diabetic foot, fat-pad infiltration may play a role in their pathogenesis.

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