Volumetric assessment of type-1 diabetic neural atrophy using voxel based morphometry

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Objective: Diabetes mellitus is a chronic multisystem disease, in which poor glycaemic control leads to a myriad of complications within the human body. Type-1 diabetic health concerns are often linked to aspects of kidney function, retinopathy, heart disease, neuropathy, et cetera. Previous work from our group has identified involvement of the central nervous system in diabetic neuropathy (1,2). Recent research has suggested an association between type-1 diabetes and brain parenchymal atrophy and possible cognitive decline (3). Such atrophy, whilst being qualitatively consistent with the normal ageing process, is manifesting itself in diabetic patients of a far younger age than would be ordinarily anticipated. The aim of this work is to quantitate any intracranial volumetric differences in a cohort of patients at various stages of type-1 diabetes-associated disease.

Methodology: Eighty-seven male subjects with type-1 diabetes underwent imaging at 1.5T (Eclipse, Philips Medical Systems, Cleveland, Ohio). Three subject groups were determined by their neuropathic status, indicative of various stages in the disease process. Classification was based on Dyck’s neuropathy staging criteria (4): The 3 groups comprised 30 sub-clinical, 29 painful and 28 patients with painless neuropathy. Three-dimensional, T1-weighted image datasets (TE=4.4ms; TR=15ms) were acquired using a radiofrequency-spoiled Fast Acquisition in the Steady-State sequence (RF-FAST). Whole-head coverage was obtained in all cases with an isotropic voxel resolution of 1mm*1mm*1mm. Data was pre-processed and compared using Statistical Parametric Mapping (SPM2) Voxel Based Morphometry.

Results: The sub-clinical neuropathy group demonstrated greater grey matter probability density than both painful and painless groups (P<0.001, uncorrected). This occurred primarily in the occipital cortex when compared to the painless group and was more generalized over the entire cortical structures when compared to the painful group (Fig. 1). White matter was also greater in the sub-clinical neuropathy group and cortical CSF was greater in the painful and painless groups when compared to the sub-clinical neuropathy group.

Discussion: These results are consistent with increases in CSF plus concomitant decreases in both grey and white matter in those with clinically-apparent diabetic neuropathy. Different patterns of atrophy were identified at different disease stages.

References: