High resolution, quantitative magnetic susceptibility and phase mapping MRI techniques show increased iron…

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Introduction: Multiple sclerosis (MS) is an autoimmune disease of the central nervous system leading to demyelination and axonal loss. Oligodendrocytes and myelin are rich in iron and neurodegeneration in MS might be expected to cause an accumulation of iron in deep grey matter (dGM) structures which have extensive expression of the transferrin receptor and the ferrous iron transporter. The change in magnetic susceptibility due to iron accumulation causes local field shifts which can be detected on phase images particularly at high field MRI, which can be inverted to produce maps of the distribution of magnetic susceptibility. The aim of this study is to measure the magnetic susceptibility and phase values of dGm structures in patients with relapsing-remitting MS (RRMS) patients and correlate them with the T2 lesion load, Extended Disability Status Scale (EDSS), number of relapses, and disease duration.

Methods:

Subjects: 30 RRMS patients were recruited from the neurology clinic at King Hussein Medical Centre, Jordan along with 30 age and sex matched healthy controls. All participants were consented. Clinical and EDSS assessments were carried out a senior neurologist.

MR Imaging: Scanning was performed on a 3T Siemens Trio MR system. High resolution 3D T2*-weighted gradient echo and T2-weighted Fluid-Attenuated Inversion Recovery (FLAIR) sequences were obtained. Both magnitude and phase images of the T2* sequence were reconstructed for further analysis.

Post Processing and Analysis:

Phase images were converted into susceptibility maps on which different regions-of-interest (ROIs) were drawn to cover the caudate nucleus (CN), putamen (PT), globus pallidus (GP) and thalamus (TH). The posterior horn of internal capsule was used as a normal reference value. T2-weighted FLAIR images were used to calculate the lesion load.

Results: For the RRMS patients, significant moderate correlation ($p < 0.05$) was found between the EDSS and the susceptibility in both CN ($r=0.522$) and PT ($r=0.532$). Significant differences ($p < 0.05$) in magnetic susceptibility were found between RRMS and HC subjects for the PT, GP and TH. Furthermore, significant differences ($p < 0.05$) in phase values were found between RRMS and HC subjects for the CN, PT, GP, and TH.

Conclusion: High resolution susceptibility and phase mapping techniques were found to be sensitive to changes in iron deposition, and showed increased iron deposition in RRMS patients compared to HCs.

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Increased total sodium concentration in asymptomatic T2 lesions in clinically isolated syndrome

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Background: Increased total sodium concentration has been demonstrated in the brains of patients with relapsing remitting, secondary progressive and primary progressive multiple sclerosis (MS). It is thought that this may reflect neuroaxonal pathology. However, it is still unknown if increased sodium is seen in subjects presenting with a clinically isolated syndrome (CIS).

Objective: The aim of this study is to estimate the total sodium concentration in the normal appearing white matter, cortical grey matter, deep grey matter and T2 lesions in patients presenting with a CIS.

Methods: We recruited 20 CIS subjects (10F:10M), with a mean age 35.6 (±8.6) years within 3 months from symptom onset and 11 healthy controls (7F:4M), with a mean age 35.2 (±7.5) years. Patients had a median EDSS of 1.0 (range 0-2.5), with the majority of subjects presenting with an optic neuritis (N=17). 11 of the CIS subjects had an abnormal MRI scan with asymptomatic, non-enhancing T2 lesions at baseline (mean T2 lesion volume = 6.20 (±5.5) ml). Subjects underwent two protocols in the same session using a 3T scanner: 1) PD/T2 and 3D T1 images; and 2) quantitative total sodium MRI. After lesion filling the 3D T1 images, probabilistic tissue brain segmentations were performed using GIF. Tissue masks were registered and resampled to the sodium space using NiftyReg. Linear regression models compared differences between groups adjusting for age, gender and grey or white matter tissue fraction.

Results: Increased sodium concentration was seen in the asymptomatic brain T2 lesions compared with normal appearing white matter in CIS subjects (49.98±7.77mM versus 33.16±3.7mM, $p=0.005$). There were no differences in sodium concentration for normal appearing white matter (33.16±3.7mM versus 31.4±2.3mM), cortical grey matter (41.8±3.2mM versus 41.1±2.6mM) and deep grey matter (35.5±3.3mM versus 34.4±2.5mM) in CIS patients when compared to controls.

Conclusion: This study extends the findings of increased sodium concentration in MS to patients presenting with a CIS. Increased total sodium levels in the T2 lesions could reflect the underlying oedema, associated with an inflammatory event; additionally, this could reflect neuroaxonal dysfunction and/or loss occurring at the onset of a first demyelinating event. Sodium imaging may have a role as an outcome measure in clinical trials that target the sodium pathway of the neurons.

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