normal with concomitant suppression of TSH. In 2 of the 4 cases, the hyperthyroidism resolved within 4-8 weeks with no antithyroid treatment, and subsequently the patients became permanently hypothyroid. In one case, treatment with antithyroid medication was discontinued after 4 months when the patient became hypothyroid. Only in one case was there evidence of sustained hyperthyroidism requiring continuous treatment with antithyroid medication methimazole for one year with good response.

CONCLUSIONS: In this small case series, the majority of patients developing hyperthyroidism following treatment with alemtuzumab have a temporary course of hyperthyroidism pathognomonic of autoimmune thyroiditis which resolves quickly with minimal medical treatment. In the case of Graves' disease induced by alemtuzumab, conservative management with methimazole rather than aggressive treatment with surgery or RAI treatment is shown to be both efficacious and feasible.

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Keywords: Disease-modifying treatments in MS, Immunology and MS

(P35)

CHEMICAL EXCHANGE SATURATION TRANSFER IMAGING IN MULTIPLE SCLEROSIS

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BACKGROUND: Chemical exchange saturation transfer (CEST) is a new MRI technique that uses saturation (signal destruction) to detect exchangeable protons (such as in OH, NH groups) in very low concentration solutes (mM) via the water signal in MRI. Previous work showed that CEST is related to mobile proteins. The technique is similar to a traditional magnetization transfer (MT) pulse sequence but uses longer duration and low power pulses with minimal effect on MT. In order to assess multiple exchangeable pools, saturation pulses are applied at a number of offset frequencies relative to water, and the free water signal is quantified.

OBJECTIVES: To apply a new CEST MRI acquisition and data processing technique to MS patients that has minimal MT contribution, and to quantify the CEST-NOE (nuclear overhauser effect).

METHODS: Patients with multiple sclerosis were scanned on a 3T MRI scanner. The CEST acquisition consisted of a 3D segmented-EPI readout with a 7 shot EPI. Each excitation was preceded by a 25 ms, 1μT saturation pulse. The volume acquisition time was approximately 11s. Volumes were acquired with the saturation pulse frequency swept from -10 to 10 ppm in 65 steps. For each voxel, a subset of the z-spectrum was fit to a Lorentzian function and the Lorentzian difference (LD) was calculated. MPRAGE, T2-weighted, and FLAIR images were also acquired as part of the imaging protocol.

RESULTS: Two female, secondary progressive MS patients have been analyzed. The first was a 58 year old female who had a single large periventricular lesion that was hyperintense on both T2 and FLAIR. The corresponding CEST-NOE image showed a hypointensity of approximately the same size. The second was a 55 year old who had multiple lesions, both periventricularly and in the deep white matter. Several lesions showed the same pattern to the first patient in that the lesion was hyperintense on T2 and FLAIR and hypointense on CEST-NOE. Another lesion was hyperintense on T2 and FLAIR, but the hypointensity was less than half the size on the CEST-NOE image. Another frontal, periventricular lesion was hyperintense on T2, but was isointense on FLAIR and hypointense on CEST-NOE.

CONCLUSIONS: These results suggest the CEST-NOE image has contrast different from T2 and FLAIR images. Due to the low power saturation pulse, it is not likely to be MT dependant, and therefore might show novel protein changes compared to standard MRI sequences.

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Keywords: Imaging and MS

(P36)

INTERFERON-BETA REDUCES MITOCHONDRIAL DISTRESS IN EAE

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BACKGROUND: Inflammatory tissue injury is the hallmark of multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE). This damage includes neuronal loss and recent observations suggest that oxidative stress and mitochondrial dysfunction play an important role in neuronal injury. Mitochondria are dynamic organelles that respond to oxidative stress by undergoing fission and fusion. Interferon-beta (IFN-β) is a pleotropic cytokine which is widely used in the treatment of MS and is thought to act as an immunomodulator. Some evidence supports the ability of IFN-β treatment to reduce neuronal loss in MS but little is known about the mechanisms that might underlie this effect.

OBJECTIVES: The aim of this study was to examine the effect of IFN-β on mitochondrial dysfunction in myelin oligodendroglial peptide-induced EAE in C57Bl/6J mice.

METHODS: EAE was induced with MOG peptide 35-55, genetically engineered bone marrow stem cells were used to deliver IFN-β and tissues were examined immunohistochemically as previously described (Makar, et al, J Neurol Sci 2008; 196: 67).

RESULTS: We showed for the first time that the mitochondrial fission protein Fis1, is increased and the mitochondrial fusion protein, Mrf2, is decreased in this EAE model. This correlated with increases in Cytochrome C. EAE mice transplanted with