Proton MR Spectroscopy Findings in Chronic Neuroborreliosis

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Introduction

Chronic Lyme disease (CLD) patients frequently complain of continuing neurological symptoms even after receiving the antibiotic therapy recommended by the Infectious Disease Society of America (1). Several clinical trials have shown no benefit to prolonged antibiotic therapy, causing many to question not only whether CLD represents ongoing infection, but also whether there is an underlying pathology or is purely psychiatric (2). As neuroimaging of CNS Lyme or Neuroborreliosis rarely demonstrates any abnormality even in the acute phase, this has been difficult to study. Proton MR Spectroscopy (1H-MRS) is a potentially powerful, yet underutilized, tool to explore neurological dysfunction via the quantification of several metabolites that indicate neuronal health, inflammation or demyelination. To date there is only one preliminary study of MRS findings in CNS Lyme and no MRS studies focusing on CLD(3). Our goal is to use MRS to examine patients diagnosed with CLD for objective findings of underlying neuropathology.

Methods and Materials

Three patients (2 females 18 and 38 years old, 1 male 44 years old) being treated for CLD were imaged at 3T with T1 MP-RAGE and T2 weighted MRI. N-acetyl aspartate (NAA), choline (Cho), creatine (Cr) and *myo*-inositol (mI) were quantified within a 360ml volume of interest (VOI) with 3D 1H-MRSI and the NAA was also quantified globally with a whole-brain NAA (WBNAA) 1H-MRS sequence (4).

Results

MRI of patient 3 revealed two foci of nonspecific T2/FLAIR hyperintensity. No abnormalities were seen on MRI of patients 1 and 2. WBNAA was significantly decreased for patients 1 and 2 but was normal for patient 3. Choline was significantly increased for patient 3 and in the low normal range for patients 1 and 2. All other markers examined were within the normal range (see Fig. 1).

Discussion

NAA, a marker of neuronal health and membrane integrity (4) was decreased in patients 1 and 2, consistent with underlying pathogenesis. Since all patients had normal NAA in their VOI, the decreased WBNAA likely represents gray matter dysfunction. However, the increased choline is consistent with an inflammatory or demyelinating process (5). While none of the abnormalities noted are specific to spirochetal infection or any other neuropathology, they do indicate an underlying pathological process beyond psychiatric disorders.

Conclusion

Patients with CLD are likely to have underlying CNS pathology and greater effort should be made to determine and treat these underlying causes rather than dismissing symptoms as a psychiatric syndrome.

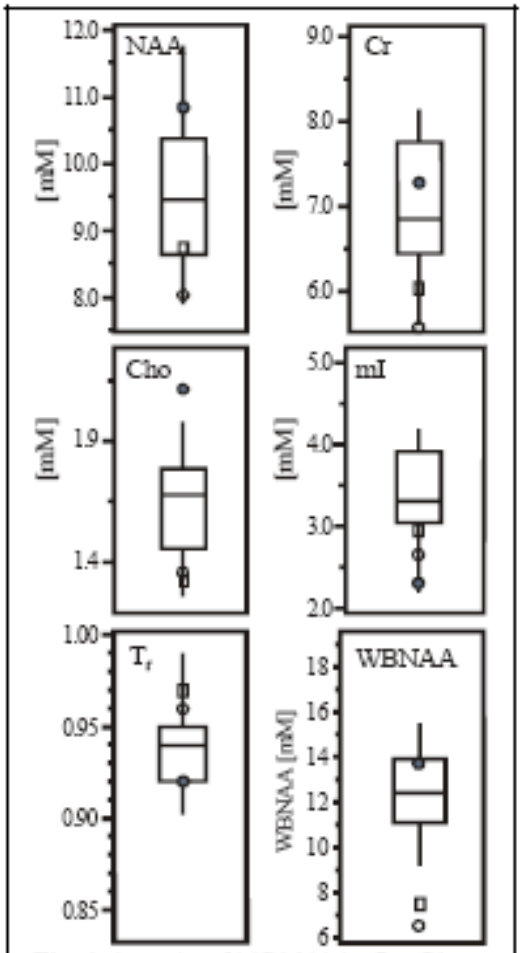

Fig 1: Levels of VOI NAA, Cr, Cho, mI and tissue fraction (Tf) and WBNAA in patients 1 (○), 2 (■) and 3 (●) over corresponding values from 101 healthy controls.

Reference

1. Wormeser et al. CID 2006:43(1):1089-1134
2. Hassett et al. Arthritis Rheum 2008(12):1742-1749
3. Ustymowicz et al. Neruoradiology 2004(46):26-30
4. Rigotti et al. Am J Neuroradiol 2007(28):1843-1849
5. Rosen et al. Neurotheraputics. 2007(4):330-345